In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 17-1378V

Filed: November 13, 2023

JODI FISKE, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	*	Special Master Katherine E. Oler

Andrew Downing, Downing, Allison & Jorgenson, Phoenix, AZ, for Petitioner
Katherine Edwards, U.S. Department of Justice, Washington, DC, for Respondent

RULING ON ENTITLEMENT[1]

On September 28, 2017, Jodi Fiske ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program[2] alleging that she suffered small fiber neuropathy ("SFN") as a result of the influenza ("flu") vaccination she received on September 29, 2014. Pet. at 1, ECF No. 1 at 2. For the reasons set forth below, I find that Petitioner has preponderantly demonstrated that the flu vaccine caused her condition. She is therefore entitled to compensation.

[1] Because this Ruling contains a reasoned explanation for the action in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Ruling will be available to anyone with access to the internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) ("Vaccine Act" or "the Act"). Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

I. Procedural History

Petitioner filed her petition on September 28, 2017. ECF No. 1. She filed medical records on October 17, 2017 (Exs. 1-5), November 22, 2017 (Exs. 6-9), January 15, 2018 (Exs. 10, 11), and February 26, 2018 (Ex. 12). She filed a statement of completion on February 26, 2018. ECF No. 17.

Respondent filed his Rule 4 report on August 27, 2018, stating that the case was not appropriate for compensation under the terms of the Vaccine Act. ECF No. 23.

For the next year, Petitioner and Respondent filed reports and supporting medical literature from their respective experts. Exs. 14, 37 (expert reports from Petitioner's expert, Dr. Laura Boylan); Exs. A, C (expert reports from Respondent's expert Dr. Christopher Gibbons).

On July 28, 2020, Petitioner indicated that she did not intend to submit any additional expert reports and asked that her case be scheduled for an entitlement hearing. ECF No. 50. Based on the parties' submissions, I scheduled the entitlement hearing for October 20, 2021. *See* Non-PDF Scheduling Order dated August 26, 2020.

Petitioner filed her pre-hearing brief on September 22, 2021. ECF No. 54. Respondent filed his pre-hearing brief on September 29, 2021. ECF No. 57. Petitioner filed updated medical records on October 6, 2021. Ex. 57. Petitioner filed a pre-hearing reply brief on October 6, 2021. ECF No. 61.

I held an entitlement hearing via Zoom on October 20, 2021. After that, the parties filed post hearing briefs. This case is ripe for an adjudication.

II. Medical Terminology

Neuropathy refers to nerve damage in the peripheral nervous system. Tr. at 133. The nervous system has multiple components: large nerve fibers coated in myelin, unmyelinated small nerve fibers that control autonomic function, and lightly myelinated autonomic nerve fibers, which are often classified as small fibers. *Id.* The autonomic nervous system controls involuntary functions such as respiration, heart rate, blood pressure, sweating, and digestion. *Id.* at 134.

Small fiber neuropathies are disorders that damage thinly myelinated and unmyelinated nerve fibers. Terkelsen et al., *The diagnostic challenges of small fibre neuropathy: clinical presentations, evaluations, and causes*, 16 LANCET NEUROL 934-44 (2017) (filed as Ex. 18) (hereinafter "Terkelsen").

SFN exists in length dependent and non-length dependent forms, where length refers to the length of the nerve. Tr. at 69. Length dependent SFN normally begins in the patient's feet and ascends to the hands. *Id.* at 167. In some cases, SFN occurs in non-length dependent fashion, meaning that it begins elsewhere in the body and moves to different locations in non-linear distribution. *Id.* at 168. Terkelsen depicts the length-dependent and non-length-dependent forms

of SFN below.

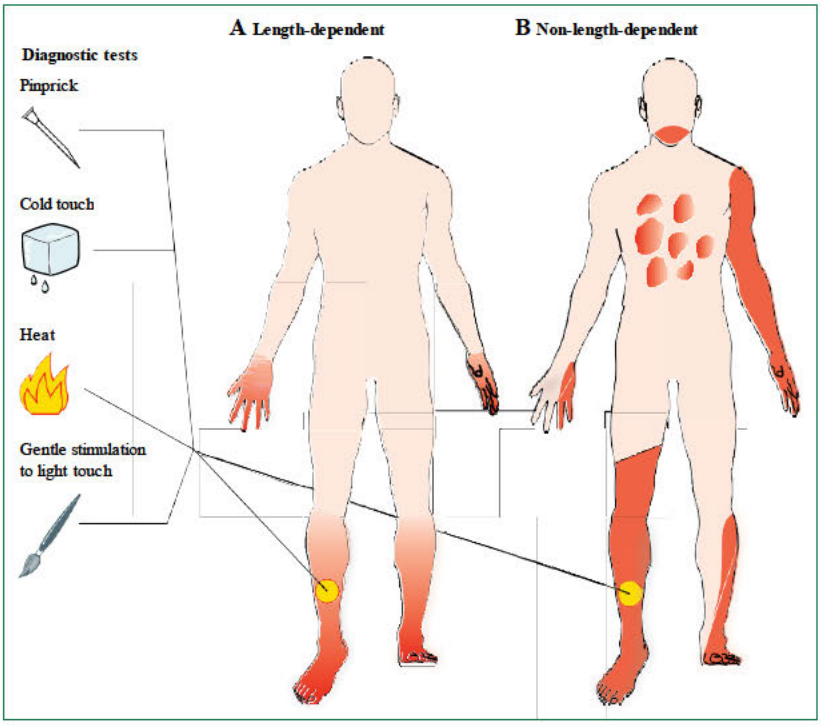

Figure 1: Clinical presentations in small fibre neuropathy (SFN)
A patient with typical length-dependent polyneuropathy (A) might have pain, sensory loss, or hypersensitivity to cold, warm, light touch, or pinprick in a characteristic stocking-glove distribution, with intact deep tendon reflexes and preserved proprioception and sensation to vibration. A patient with patchy non-length-dependent neuropathy
(B) might have either reduced or increased small fibre function corresponding to a single or to multiple nerves.

Terkelsen at 935.

III. Medical History

A. Pre-Vaccination History

Petitioner's pre-vaccination medical history is remarkable in that she suffered a peroneal[3] nerve injury in her left knee in 2002. Ex. 2 at 7. She experienced numbness and tingling in the distribution of the left peroneal nerve but no motor weakness or foot drop. *Id.* The tingling receded, but the numbness was persistent. *Id.*

On May 18, 2011, Petitioner was experiencing facial numbness and underwent a brain MRI. Ex. 9 at 10. The MRI revealed maxillary sinus disease without acute features, but no other abnormalities. *Id.*

On November 7, 2012, Petitioner visited Zaib Ukani, MD, her primary care provider, for shoulder pain and muscle pain in the upper left of her back. Ex. 8 at 9. The pain persisted, and on November 21, 2012, a right shoulder x-ray was performed and did not reveal any abnormalities. Ex. 9 at 21.

[3] Peroneal: "pertaining to the fibula or to the lateral aspect of the leg." www.dorlandsonline.com/dorland/definition?id=38138&searchterm=peroneal (last accessed Oct. 26, 2023).

On December 26, 2012, Petitioner underwent an MRI of her cervical and thoracic spine. Ex. 9 at 29-30. The reason for the MRI is listed simply as "pain." *Id.* at 28. The cervical spine MRI revealed a diffusely enlarged thyroid gland and a "[n]egative study of the cervical spine." *Id.* at 29. The MRI of Petitioner's thoracic spine revealed "posterior lateral right chest wall clinical marker at the T5 level immediately medial to the right scapula overlies an interval healing posterior lateral right 4th rib fracture, this only seen on the transverse images." *Id.* at 30. It found no other significant abnormalities. *Id.*

Petitioner received the allegedly causal flu vaccine in her left deltoid on September 29, 2014, at the age of 36. Ex. 1 at 1. There had been no recent changes in symptoms of her peroneal nerve injury at the time of Petitioner's vaccination. Ex. 2 at 7.

B. Post-Vaccination History

On November 4, 2014, Petitioner saw Dr. Ukani for joint and muscle pain for five weeks, generalized aches and pains for a year, and feeling stressed. Ex. 8 at 5. Her examination and labs returned no abnormal results. *Id.* Dr. Ukani's assessment was myalgia and chronic SSRI use. *Id.* He increased the dosage of Petitioner's Zoloft prescription. *Id.*

On November 12, 2014, Petitioner saw neurologist Frank Urban, MD. Ex. 7 at 36-41. The record notes that she experienced numbness and tingling over the preceding six weeks as well as lightheadedness, constipation, heartburn, and dry mouth.[4] *Id.* Petitioner underwent an electromyography (EMG) nerve conduction study (NCS), but the results were not interpreted. *Id.* at 17-22. The following day, Petitioner underwent a brain MRI which showed no abnormalities and no significant changes from her MRI on December 10, 2004.[5] *Id.* at 24. A serum protein test on November 14, 2014, was also normal. *Id.* at 28.

On November 20, 2014, Petitioner visited rheumatologist David Alboukrek, MD, FACR, for arthralgia,[6] myalgia,[7] and tingling in her hands and feet. Ex. 4 at 9. Petitioner described that her neurological symptoms had worsened since her appointment with Dr. Urban on November 12, and she felt constant burning in her feet. *Id.* She also reported that her symptoms began about five days after she received the flu vaccine on September 29. *Id.* Dr. Alboukrek ordered laboratory tests which showed a negative antinuclear antibody ("ANA")[8] result and a borderline positive result for

[4] The handwritten notes documenting this appointment are difficult to interpret.

[5] The records appear to contain a transcription error which lists the date of this MRI as December 2, 2014. Ex. 7 at 23; Ex. 3 at 11.

[6] Arthralgia is "pain in a joint." DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=4230 (last visited May 23, 2023) ("Dorland's").

[7] Myalgia is "pain in a muscle or muscles." DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id =32592 (last visited May 23, 2023).

[8] Antinuclear antibodies are "antibodies directed against nuclear antigens; ones against a variety of different antigens are almost invariably found in systemic lupus erythematosus and are frequently found in rheumatoid arthritis, scleroderma (systemic sclerosis), Sjögren syndrome, and mixed connective tissue

anti-DSDNA antibodies.[9] *Id.* at 10. He indicated the DSDNA result was not clinically significant in the context of the other serologies. *Id.* Petitioner's examination was normal except for decreased pinprick sensation in her feet. *Id.* She was prescribed Voltaren and advised to return for a follow up visit in four weeks. *Id.* Dr. Alboukrek suspected Petitioner had "some type of reaction induced by the flu shot but there is no way to prove it." *Id.*

On December 4, 2014, Petitioner saw neurologist Richard Bailyn, MD. Ex. 2 at 7-9. She described pain in her forearms and tingling in all of her fingers, both beginning five days after the flu vaccination, and unresolved though gradually improving. *Id.* at 7. Three weeks before, Petitioner felt that her knees were giving way and subsequently developed painful burning and tingling in her feet and occasional unsteady gait. *Id.* She also felt occasional dizziness. *Id.* Dr. Bailyn noted that Petitioner had persistent numbness in the distribution of her left peroneal nerve with no recent change. *Id.* Sensory examination revealed hypalgesia[10] in the distribution of the left peroneal nerve but was otherwise normal. *Id.* at 8. Her neurological examination was normal except for very slight atrophy of the left anterior tibialis muscle. *Id.* Dr. Bailyn's impression was limb dysesthesia[11] of uncertain etiology and no clinical indication of generalized peripheral nerve dysfunction. *Id.* He ordered the EMG be repeated and suggested that Petitioner undergo a course of physical therapy ("PT"). Ex. 2 at 8.

On December 18, 2014, Petitioner saw Dr. Alboukrek for a follow-up. Ex. 4 at 7-8. Petitioner reported that her arthralgias had partially improved and that she never took the prescribed Voltaren. *Id.* at 7. Her neurological symptoms were still present but had also improved. *Id.* Petitioner also developed a rash on her left arm and abdomen. *Id.* Petitioner had decreased pinprick sensation in her feet. *Id.* at 8. Records note that the results of the November 20 laboratory tests were normal "except for the borderline DSDNA." *Id.* Dr. Alboukrek ordered the testing be repeated. *Id.* at 7. On December 19, 2014, Petitioner's DSDNA test was equivocal. *Id.* at 12.

On January 13, 2015, Petitioner saw neurologist Khema Sharma, MD. Ex. 6 at 1-3. Petitioner complained of numbness, tingling, and pain with acute onset after her September 29, 2014, flu vaccination. *Id.* at 1. Petitioner reported that five days after the vaccination she noticed burning pain, numbness, and impaired dexterity in both hands and forearms. *Id.* She noted burning pain in her feet which ascended to her legs in the first week of November 2014. *Id.* Petitioner also experienced weakness in her legs from the same period, describing feeling as though her legs were going to give out, difficulty walking for more than five minutes, and difficulty standing up. *Id.* Dr.

---

disease." DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id=56804 (last visited on May 23, 2023).

[9] Anti-DSDNA antibodies are "a type of antinuclear antibody specific for double-stranded DNA, found in the serum of patients with systemic lupus erythematosus." DORLAND'S, https://www.dorlandsonline.com /Dorland/definition?id =56790 (last visited May 23, 2023).

[10] Hypalgesia is "decreased nociception (pain sense)." DORLAND'S, https://www.dorlandsonline.com/ dorland/definition?id=23626 (last visited May 23, 2023).

[11] Dysesthesia is "distortion of any sense, especially of that of touch," or "an unpleasant abnormal sensation produced by normal stimuli." DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id=15186 (last visited May 23, 2023).

Sharma noted that Petitioner's brain MRI, EMG, nerve conduction study, laboratory tests, and repeated DSDNA testing were normal. *Id.* at 2. Petitioner's symptoms improved over the three to four weeks preceding the January 13 appointment and the rash she had reported on December 18 had resolved. *Id.* Sensory examination revealed impaired light touch and pinprick sensation in the median nerve distribution in both hands and distally in the feet. *Id.* at 3. Strength examination found mild weakness in her toe extensors. *Id.* at 2-3. Dr. Sharma also noted that Petitioner sensed vibration for six seconds on both sides and exhibited mild impairment of joint position sense on her right great toe. *Id.* at 3. Dr. Sharma assessed that Petitioner's symptoms suggested small fiber dysfunction, likely due to a post-infectious process as "all the possible causes of the small fiber neuropathy are negative". *Id.* at 3. Dr. Sharma also noted that other potential explanations such as pyramidal tract dysfunction of the posterior column, cervical myelopathy, and chronic use of SSRI's such as Zoloft which Petitioner had taken for an extended period, could not be ruled out. *Id*. Dr. Sharma ordered an MRI of the cervical spine, which took place on January 30 and showed no abnormalities. Ex. 3 at 7.

On February 9, 2015, Petitioner saw orthopedic surgeon Jeffrey Fernyhough, MD, for back pain following a February 2, 2015, motor vehicle accident. Ex. 8 at 19-21. Petitioner's symptoms of numbness, tingling, and weakness in the lower legs that arose after her flu vaccination were unchanged. *Id.* at 19. Since the accident, Petitioner had stopped running and attending spinning classes due to back pain. *Id.* Dr. Fernyhough noted that Petitioner was diagnosed with "apparent small fiber neuropathy" by Dr. Sharma, although Dr. Bailyn "opined it was some other post flu vaccine neuropathy." *Id.* Dr. Fernyhough diagnosed Petitioner with posterolateral trunk pain, likely musculoskeletal or myofascial. *Id.* at 21. He also noted Petitioner had "enigmatic post vaccine neuropathy of the feet and distal legs distribution." *Id.* Dr. Fernyhough recommended Petitioner defer Advil for NSAIDs due to her "history of apparent vaccine related neuropathy syndrome" and prescribed physical therapy for her back pain. *Id.*

On February 24, 2015, Petitioner saw endocrinologist Sonia Gibson, MD, for hypoglycemia.[12] Ex. 13 at 5. Dr. Gibson noted that Petitioner started experiencing severe neuropathy and pain in the soles of her feet shortly after her flu vaccination the previous September. *Id.* Dr. Gibson also noted that a specialist found Petitioner had short fiber neuropathy. *Id.* Dr. Gibson stated that hypoglycemia could be responsible, and a four-hour glucose tolerance test was administered which showed a glucose level of 45 with no symptoms. *Id.* Petitioner's initial symptoms included hunger, palpitations, paresthesias, and tremulousness as well as lightheadedness, nausea, palpitation, sweating, tremor, and neuropathy. *Id.* The symptoms were of moderate severity, occurred weekly, and were associated with skipped meals and the postprandial[13] period. *Id.* Glucose, drinking orange juice, or eating a snack relieved the symptoms. *Id.* Petitioner did not follow a specific diet and sometimes consumed a high sugar load. *Id.* Dr. Gibson's assessment was reactive hypoglycemia and advised that Petitioner follow a diet with

[12] Hypoglycemia is "an abnormally diminished concentration of glucose in the blood, which may lead to tremulousness, cold sweat, piloerection, hypothermia, and headache; when chronic and severe it may cause central nervous system manifestations that in rare cases can even be fatal." DORLAND'S, https://www.dorlandsonline.com /dorland/definition?id=24248 (last visited May 23, 2023).

[13] Postprandial means "after a meal." DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id=40558 (last visited May 23, 2023).

complex carbohydrates and protein. *Id.* at 7. Dr. Gibson also noted that if Petitioner's neuropathy was related to glucose fluctuation, she would expect it to improve with the altered diet. *Id.*

On April 8, 2015, Petitioner returned to Dr. Alboukrek for a follow-up. Ex. 4 at 5-6. Petitioner reported generalized arthralgias and myalgias and that she felt achier than on her December 18, 2014, visit. *Id.* at 5. Petitioner was also experiencing depression. *Id.* Examination revealed multiple tender points. *Id.* at 6. Dr. Alboukrek's assessment was that Petitioner's "clinical picture is becoming more suggestive of fibromyalgia syndrome." *Id.* She was advised to begin walking on a treadmill, to take Pamelor and Zoloft, and to see a psychologist for her depression. *Id.* at 6.

On May 27, 2015, Petitioner returned to Dr. Gibson. Ex. 13 at 8-10. Dr. Gibson noted that on Petitioner's February 24, 2015, lab results for cortisol, IGF1, insulin, and C-peptide were normal. *Id.* at 8. Petitioner's hypoglycemia usually occurred during the day, was not constant, and was never associated with fasting. *Id.* She changed her diet to small frequent meals with complex carbohydrates, but the symptoms continued sporadically. *Id.* Petitioner's neuropathy was not improving. *Id.* Dr. Gibson's first assessment was mild reactive hypoglycemia, for which Petitioner "does not really have any symptoms" and which was being controlled with diet. *Id.* at 9. The second assessment was unspecified idiopathic peripheral neuropathy. *Id.* Dr. Gibson noted that she did "not see an endocrine cause for her symptoms of neuropathy." She also described Petitioner's neuropathy and reactive hypoglycemia as unrelated. *Id.* Dr. Gibson recommended Petitioner try vitamin B12 injections for her hypoglycemia. *Id.* at 9-10.

On June 4, 2015, Petitioner saw physiatrist Kathleen Davenport, MD, for bilateral leg pain. Ex. 3 at 13-15. Petitioner described burning and tingling in her hands and arms which progressed to her legs, beginning five days after her September 2014 flu vaccination. *Id.* This initially had a minimal impact on her gait and balance, but eventually progressed such that she was not able to stand for more than five minutes. *Id.* Petitioner also reported leg pain extending into her ankles and feet. *Id.* She described her pain as constant with numbness, tingling, and burning and noted that it was exacerbated with exercise and improved with rest. *Id.* Petitioner also described her weakness as improved. *Id.* Petitioner estimated her current pain level as 2/10 or 3/10 but said it ranged between 1/10 or as high as 6/10. *Id.* Overall, Petitioner felt better than at the onset of her condition in October 2014, but it was still "significantly bothersome" and limited her ability to function. *Id.* Dr. Davenport's impression was "that this does appear to be [] consistent with mild Guillain Barre Syndrome based on the presentation of this. A small fiber neuropathy would also be a reasonable consideration. I am concerned about some sort of underlying infectious etiology given the occurrence shortly after the flu shot and with a history of rash at some point." *Id.* at 14. She recommended a lumbar and thoracic spine MRI, a repeat EMG, and a Lyme titer. *Id.* Petitioner did not wish to pursue treatment without a diagnosis. *Id.*

On August 13, 2015, Petitioner saw podiatrist Jodi Schoenhaus, MD, for pain and burning in her feet. Ex. 10 at 5-6. Petitioner relayed that her symptoms began after her September 2014 flu vaccination. *Id.* Examination found that Tinel sign[14] was present on the left and right posterior

[14] Tinel sign is "a tingling sensation in the distal end of a limb when percussion is made over the site of a divided nerve. It indicates a partial lesion or the beginning regeneration of the nerve." DORLAND'S MEDICAL DICTIONARY ONLINE (hereinafter "DORLAND'S"), https://www.dorlandsonline.com/dorland/

tibial nerves and the left and right superficial peroneal nerve. *Id.* at 6. Dr. Schoenhaus's assessment was neuralgia, neuritis, and radiculitis. *Id.* Dr. Schoenhaus discussed the causes and etiology of peripheral neuropathy with Petitioner and prescribed Lyrica. *Id.*

On August 17, 2015, Petitioner underwent a thoracic spine MRI, which was normal. Ex. 3 at 3. A lumbar spine MRI performed on the same day showed a herniated disc at the L2-3 and L3-4 levels and two cysts of unclear etiology for which a CT scan and pelvic ultrasound were suggested. *Id.* at 4-5.

On September 8, 2015, Dr. Alboukrek provided Petitioner with an exemption from the seasonal flu vaccine because she had a "severe allergic reaction" to it the previous year. Ex. 12 at 8. On October 5, 2015, Petitioner declined the flu vaccine. *Id.* at 7.

On January 20, 2016, Petitioner saw her PCP for a medication refill. She reported that she had a reaction to the flu shot with numbness and tingling. Ex. 8 at 4.

On September 19, 2016, Petitioner saw Dr. Gibson for neuropathy and hypoglycemia. Ex. 13 at 11. She continued to experience intermittent burning and numbness, with nocturnal paresthesias, restlessness, and tingling in both legs and the soles of both feet. *Id.* Dr. Gibson also noted that Petitioner had been evaluated by three neurologists and diagnosed with idiopathic neuropathy but was not placed on a course of treatment. *Id.* Petitioner felt better on the B12 injections but continued to have occasional flares. *Id.* She also continued to experience symptoms of reactive hypoglycemia, frequently after breakfast. *Id.* When experiencing reactive hypoglycemia symptoms, Petitioner's blood sugar was "found to be 40s." *Id.* Dr. Gibson's assessment was "other hypoglycemia" for which she recommended eating small frequent meals that include protein and avoiding complex carbohydrates. *Id.* at 12. Dr. Gibson's assessment was also a peripheral nervous system disorder. *Id.* Petitioner was to be screened for MGUS (monoclonal gammopathy of undetermined significance) and started on Metanx (a prescription food product for treatment of neuropathy). *Id.*

On October 21, 2016, Petitioner visited Dr. Ukani, who noted that she needed an influenza vaccine exemption due to her neuropathy following vaccination. Ex. 8 at 3. The neuropathy in Petitioner's arms had improved, but it remained present in her legs. *Id.* Dr. Ukani's assessment was "periph[eral] neuropathy post Flu shot." *Id.*

On August 21, 2017, Petitioner saw neurologist Jennifer Buczyner, MD. Ex. 11 at 7. An examination showed normal strength and sensation, and reduced reflexes. *Id.* at 11. Petitioner's EMG was normal. *Id.* at 12. She underwent a skin biopsy. *Id.* at 8. The assessment was neuropathy "related to either post infectious or exposure to influenza vaccine which sounds temporally related. Unfortunately, it is significantly difficult to prove causation and advised her she likely will not ever know for certain." *Id.* She had persistent sensory complaints which might be SFN or CIDP. *Id.* Dr. Buczyner ordered an EMG, which was normal. *Id.* at 12. Petitioner's skin biopsy was consistent with small fiber neuropathy. Ex. 8 at 63, 66.

---

definition?id=106510 (last visited May 23, 2023).

On January 24, 2018, Petitioner returned to Dr. Buczyner because her neuropathy had worsened. Ex. 57 at 1. Dr. Buczyner's assessment was SFN and she noted that Petitioner had an abnormal skin biopsy result consistent with that diagnosis. *Id.* at 2.

IV. Petitioner's Testimony

Petitioner testified at the entitlement hearing. Tr. at 8-24, 254-56. Prior to her September 29, 2014, flu vaccination, Petitioner testified that she was doing well. *Id.* at 8. She worked as an occupational therapist, a physically demanding job involving transferring patients on and off beds and into chairs. *Id.* at 8-9. Prior to vaccination Petitioner suffered from anxiety but described it as "very well controlled." *Id*. at 9.

Petitioner testified that she was diagnosed with a superficial peroneal nerve injury in 2002. Tr. at 9. At the time, Petitioner was frequently on her knees while working at a pediatric therapy clinic. *Id*. Petitioner saw a neurologist who thought the injury was due to compression of her knees. *Id*. She experienced numbness extending from the left knee down to the left ankle, but after receiving physical therapy the symptoms resolved. *Id*. at 9-10. She was not experiencing symptoms at the time of the September 29, 2014 vaccination. *Id*. at 10.

Petitioner stated that she felt normal on the day of her vaccination, with no signs of infection, headache, nausea, fatigue, fever, or other health complaints. Tr. at 10. Approximately five days later, Petitioner began to experience tingling and numbness in both feet, and a burning sensation that spread into her legs, arms, and hands over the following day. *Id*. at 11. Petitioner also experienced fatigue and heaviness in her extremities. *Id*. She reported her symptoms to the employee health nurse where she received the vaccination and was advised to follow up with her regular physician if the symptoms did not resolve within the following weeks. *Id*. This visit to the employee health nurse is not included in Petitioner's records. *Id*. at 12.

Petitioner testified that her symptoms continued to worsen in October 2014 as the numbness, burning, and tingling progressed, and she felt pain and discomfort in all her extremities. Tr. at 12-13. No weakness was found on muscle testing, but Petitioner experienced the sensation of weakness. *Id*. Petitioner had difficulty performing tasks at work and around her home. *Id*. at 12.

Petitioner testified that approximately one month after vaccination, on November 4, 2014, she saw Dr. Ukani. Tr. at 13. At that time, she was experiencing burning, numbness, tingling, and fatigue through her extremities and difficulty performing tasks. *Id*. Dr. Ukani recommended that Petitioner see a neurologist. *Id*.

Petitioner testified that at present, she experiences constant numbness and tingling, though her symptoms are no longer worsening. Tr. at 15. She described experiencing sensations of pain, burning, and tingling in both legs, over the length of the entire leg, to include the thigh. Tr. at 254-55.

Petitioner testified that her doctors have recommended that she no longer receive vaccines, and so she has not been able to receive the COVID-19 vaccine. Tr. at 16. Consequently, she is unable to work in the hospital where she was previously employed. *Id*.

V. Expert Opinion and Qualifications

A. Petitioner's Expert: Laura Boylan, MD, FAAN

1. Qualifications

Dr. Boylan received her medical degree from the Columbia University College of Physicians and Surgeons in 1994. Ex. 15 ("Boylan CV") at 1. She is board certified in neurology. *Id*. at 2. Dr. Boylan currently holds the clinical positions of attending neurologist at Bellevue Hospital Center in New York City and of casual staff neurologist at St. Mary's Medical Center in Duluth, Minnesota. *Id.* at 1. Dr. Boylan is also an adjunct professor of neurology at the New York University School of Medicine where she teaches on autonomic nerve dysfunction, movement disorders, emotion in neurological diseases, and general neurology. *Id*. at 2. She has published 11 peer reviewed research reports and 69 clinical observations, reviews, and commentaries. *Id.* at 8-12. Her main research interests are behavioral neurology and neuropsychiatry, movement disorders, endogenous and exogenous brain stimulation, Parkinson's disease, brain plasticity, ethics, and human factors in patient care. *Id*. at 5. I recognized Dr. Boylan as an expert in the field of neurology. Tr. at 35.

2. Expert Reports

Dr. Boylan submitted two expert reports in this case.

a. First Expert Report

In her first expert report, Dr. Boylan noted that in rare occasions, the boost to the immune system caused by vaccination can damage the nervous system. Ex. 14 ("First Boylan Rep.") at 2. She provided two examples of pathogens that can provoke autoimmune diseases: hepatitis B can cause polyarteritis nodosa, and group A streptococcal infection can cause rheumatic fever. *Id.* at 2-3. She further noted that post-vaccine and post-infectious autoimmune conditions occur with six weeks following exposure. *Id.* at 3.

After summarizing Petitioner's medical history, Dr. Boylan noted that Guillain Barré syndrome ("GBS") has many different variants to include 12 different forms "with varying clinical and electrodiagnostic presentations." First Boylan Rep. at 8. She then stated that "[n]umerous authors have proposed that isolated small fiber neuropathy is a form of GBS." *Id.* Dr. Boylan opined that SFN can be caused by vaccination via molecular mimicry. *Id.* at 9. She defined molecular mimicry as occurring when "external agents share peptide or protein sequences with the cells which form the normal building blocks of the body." *Id.*

Dr. Boylan concluded that Petitioner "acquired an atypical form of Guillain Barre Syndrome manifesting as an isolated small fiber neuropathy as a result of her influenza vaccination of September 29, 2014." First Boylan Rep. at 10. She listed several reasons for this conclusion, to include: 1) Petitioner developed biopsy proven SFN within one week of her flu vaccine; 2) auto immune induced SFN is likely as no other cause was identified; 3) the time course of Petitioner's SFN is what one would expect for GBS, "with onset of non-specific inflammatory aches and pains

and a rash in the area of the vaccination site shortly following the vaccine with subsequent neuropathic pain in all limbs"; 4) it is common to see small fiber involvement in GBS; 5) GBS has many different phenotypes, to include one that is a sensory syndrome where there are minimal findings on EMG/NCS.

b. Second Expert Report

In her second expert report, Dr. Boylan responded to Dr. Gibbons' expert report. She began by stating that "Dr. Gibbons' sweeping statement that 'there is no evidence of small fiber neuropathy at any point in this case' is an opinion that would be far outlying among medical professionals familiar with SFN." Ex. 37 ("Second Boylan Rep.") at 2. She noted that Petitioner has "unequivocal symptoms" of pain and dysesthesias in her hands and feet and that these symptoms are characteristic of SFN. *Id.*

Dr. Boylan disagreed with Dr. Gibbons' statement that none of Petitioner's providers found sensory loss on exam. Second Boylan Rep. at 3. She stated that "sensory loss was indeed found by neurologist Dr. Sharma as well as by rheumatologist Dr. Alboukrek on two different visits." *Id.* (citing Ex. 6 at, Ex. 4 at 10). She also noted that a study found pin prick testing was normal in 30% of SFN patients. *Id.* (citing Blackmore & Siddiqi, *Pinprick Testing in Small Fiber Neuropathy: Accuracy and Pitfalls*, 17(4) CLINICAL NEUROMUSCULAR DISEASE (2016) (filed as Ex. 40)).

Dr. Boylan next disagreed with Dr. Gibbons' opinion that weakness while standing and walking is not a symptom of SFN. Second Boylan Rep. at 4. She opined that "difficulty standing an[d] walking" represents one way that a patient describes orthostatic intolerance, which can be caused by SFN. *Id.*

Dr. Boylan went on to discuss the skin biopsies in Petitioner's case, stating that "[s]kin biopsy provides a definitive diagnosis of SFN in [Petitioner]." Second Boylan Rep. at 5. She opined that "an abnormal skin biopsy almost certainly predicts SFN but a normal skin biopsy is less meaningful." *Id.* She stated that "[n]ormal testing for epidermal nerve density does not exclude SFN" because negative results are common. *Id.* at 6.

Dr. Boylan disagreed with Dr. Gibbons' contention that Petitioner's abnormal skin biopsies should be discounted because they do not square with the clinical findings, opining that "[f]indings of abnormalities are not limited to symptomatic areas." Second Boylan Rep. at 6. With respect to the sweat fiber density testing, Dr. Boylan noted that "[a]bnormality was only found in the left thigh where there were reduced numbers of epidermal nerve fibers as well." *Id.* However, Dr. Boylan noted that the testing has not been validated and that her opinions do not depend on sweat fiber density. *Id.*

Dr. Boylan agreed with Dr. Gibbons that isolated SFN can be a presentation of GBS and that such a presentation is very rare. Second Boylan Rep. at 6 (citing Ex. A at 5). Dr. Boylan cited medical literature describing twelve GBS variants including a pure sensory syndrome. *Id.* (citing Vriesendorp, Francine J., *Guillain-Barré syndrome in adults: Clinical features and diagnosis*, Up To Date (2018) (filed as Ex. 54) ("Vriesendorp")).

Dr. Boylan discussed the work of one of Dr. Gibbons' colleagues in which 155 patients with biopsy-proven SFN underwent serum testing. Second Boylan Rep. at 8-9 (citing Levine, et al., *Cryptogenic small-fiber neuropathies: Serum antibody binding to trisulfated heparan disaccharide and fibroblast growth receptor-3*, 61 MUSCLE & NERVE 512-15 (2020) (filed as Ex. 38) ("Levine")). Dr. Boylan opined that the prevalence of certain antibodies in this cohort "is strong evidence supporting a prominent role for antibody mediation in acute onset small fiber neuropathy. I find the concordance between the constellation of symptoms in the study population and [Petitioner] uncanny." *Id.* at 9.

Dr. Boylan concluded her report by opining that Petitioner "has an atypical variant of GBS manifesting as an acute onset, auto-immune mediated and non-length dependent small fiber neuropathy. [Petitioner's] illness was caused by the influenza vaccination administered to her on September 29th, 2014." Second Boylan Rep. at 9.

c. Testimony

Dr. Boylan testified at the entitlement hearing, where she estimated that she has treated "many thousands" of patients for peripheral neuropathy and over a thousand cases of acute autoimmune neurological disease with a confirmed diagnosis. Tr. at 29. She further estimated she has treated approximately 12 patients with isolated SFN, though thousands with non-isolated SFN, and hundreds of patients with confirmed GBS. *Id.* at 29-31. Dr. Boylan also assessed she has treated approximately a thousand patients with dysautonomia or some other form of orthostatic complaint. *Id.* at 32.

Dr. Boylan testified that neuropathy is a general term for disease or dysfunction of the peripheral nerves. *Id*. at 36. She explained that peripheral nerves are nerves which leave the spinal cord and move into the body, in contrast to the central nervous system which is made up of the spinal cord and brain, and the autonomic nervous system which "innervates the viscera." *Id.* However, Dr. Boylan stated that the autonomic nervous system is typically considered part of the peripheral nervous system. *Id*. In addition, neuropathies can be classified by fiber (i.e., nerve) size, distribution within the body, whether a single or multiple nerves are affected, or by etiology. *Id*.

Dr. Boylan testified that not all nerve issues can be detected by EMG and NCS. Tr. at 37-38. Indeed, small fiber neuropathy cannot be detected by EMG and NCS as those tests are only capable of detecting large nerve fibers. *Id*.

Dr. Boylan testified that small fiber neuropathy involves damage to small nerve fibers that are lightly myelinated and non-myelinated. Tr. at 38. She also explained that while SFN often accompanies large fiber neuropathy, SFN can occur in isolation, though more rarely. *Id*. at 39. In such cases of isolated SFN, Dr. Boylan identified burning, itching, extreme sensitivity to pain, crawling sensation, numbness, lack of sensation, and loss of temperature sensation as typical symptoms. *Id*. at 39-40. Dr. Boylan also explained that to test a potential SFN patient's sensory response, she would conduct a detailed sensory examination testing pin prick sensation, temperature sensation, vibration and joint position sense. *Id*. at 40. In addition, she would examine the patient's limbs for trophic[15] changes, discoloration, and swelling, particularly on the soles of

[15] "Trophic changes is a term used to describe abnormalities in the area of pain that include primarily

the feet and palms. *Id*.

Dr. Boylan opined that SFN should be considered a variant of GBS. *Id*. She explained that traditional GBS "not uncommonly" involves small fibers and, in her own clinical experience, burning dysesthesia typically accompanies GBS. Tr. at 41. Dr. Boylan agreed that "[t]he existence of sensory Guillain-Barre Syndrome has now been established beyond doubt…" *Id.* at 42 (quoting Seneviratne & Gunasekera, *Acute small fiber sensory neuropathy: another variant of Guillain-Barré syndrome?*, 72 J. NEUROLOGY, NEUROSURGERY, & PSYCHIATRY 540-42, 540 (2002) (filed as Ex. 23 and Ex. A, Tab 4) ("Seneviratne & Gunasekera"). Dr. Boylan also opined that sensory GBS is a demyelinating condition which leaves electrophysiological evidence, whereas there is no electrophysiological evidence of acute isolated SFN. *Id*. at 42-43.

Dr. Boylan cited medical literature that classifies acute sensory SFN as a type of GBS. Tr. at 44 (citing Uncini & Yuki, *Sensory Guillain-Barré syndrome and related disorders: an attempt at systematization*, MUSCLE & NERVE 464-70, 464 (2012) (filed as Ex. 25 and Ex. A, Tab 3) ("Uncini & Yuki")). She quoted Uncini & Yuki, saying "[o]n the basis of the size of fibers involved and the possible site of primary damage, we propose tentatively classifying sensory GBS and related disorders into three subtypes: acute sensory demyelinating polyneuropathy; acute sensory large-fiber axonopathy-ganglionopathy; and acute sensory small-fiber neuropathy-ganglionopathy." *Id.* at 464. Dr. Boylan testified that she believes Petitioner has acute sensory SFN which Uncini & Yuki classify as affecting small fibers. *Id*. at 468.

Dr. Boylan opined that vaccination could induce an acute isolated SFN via molecular mimicry. Tr. at 47. In this mechanism of injury, the antibodies produced in reaction to the vaccine are cross reactive and target parts of the body itself. *Id*. at 47. Dr. Boylan explained that this is the same mechanism of injury seen in cases of flu vaccine-triggered demyelinating GBS. *Id*. at 47-48.

Dr. Boylan next described the process by which a clinician would arrive at a diagnosis of acute isolated SFN. *Id*. at 48-49. The diagnostic process is based largely on the patient's clinical history and neurological and physical examinations with a potential confirmatory skin biopsy of epidermal nerve fiber density. *Id*. at 48-49. While an abnormal result can confirm SFN, Dr. Boylan clarified that an abnormal skin biopsy is not required to reach a diagnosis of SFN.[16] *Id*. at 49. However, Dr. Boylan opined that clinical context is an essential part of interpreting skin biopsy results as distinguishing true and false positive skin biopsies depends on the individual and population being tested. *Id*. at 50-51. According to Dr. Boylan, a skin biopsy is only one component in the diagnosis of SFN and a patient with an abnormal skin biopsy result but none of the characteristic symptoms could not be diagnosed with SFN on the basis of the skin biopsy alone. *Id*. at 51.

---

wasting away of the skin, tissues, or muscle, thinning of the bones, and changes in how the hair or nails grow, including thickening or thinning of hair or brittle nails." NIH, National Library of Medicine, www.ncbi.nlm.nih.gov/medgen/866865 (last visited Oct. 26, 2023).

[16] Dr. Boylan uses the terms positive and negative interchangeably with abnormal and normal, respectively, when discussing the results of Petitioner's skin biopsies. For consistency and ease of comprehension, the terms abnormal and normal are used throughout this ruling.

Dr. Boylan testified that Petitioner's superficial peroneal nerve compression injury in 2002 was unrelated to her present complaints. Tr. at 51. She also explained that, in this context, the term "superficial" indicates that the injury is close to the surface of the body, and not that the injury itself is minor. *Id*. The left superficial peroneal nerve, the subject of Petitioner's compression injury, emerges below her left knee and innervates the outside of her calf. *Id*. at 52. As such, Dr. Boylan opined that Petitioner's post-vaccination symptoms in her legs, arms, and hands cannot be explained by the left peroneal nerve injury. *Id*. at 52-53.

Dr. Boylan discussed the specifics of Petitioner's August 21, 2017, skin biopsies. *Id.* at 53 (citing Ex. 8 at 63). The biopsy of Petitioner's left thigh, found "significantly reduced Epidermal Nerve Fiber Density, consistent with small fiber neuropathy." Ex. 8 at 63. The biopsy of Petitioner's foot/calf was normal. *Id*. Dr. Boylan opined that the result of the foot/calf biopsy did not impact the SFN diagnosis because the test is more useful for ruling a diagnosis in than ruling one out. Tr. at 54-55. She opined that skin biopsies have a higher specificity than sensitivity, meaning that an abnormal result is highly likely to be accurate while a normal result is more likely to be a false negative. *Id*. at 55-56. Dr. Boylan testified that a skin biopsy is considered abnormal if the epidermal nerve fiber density value is at or below the fifth percentile. *Id*. at 56-57.

In response to questioning by the Court, Dr. Boylan testified that skin biopsies are typically done at standardized sites. Tr. at 107. She also stated that, in Petitioner's case, the biopsy sites were above the ankle and above the knee. *Id*. Dr. Boylan explained that biopsy sites are standardized to create norms on which to base percentile deviation in nerve fiber density and that the sites do not need to be symptomatic. *Id.* at 107-108. Additionally, she noted that the abnormal result from Petitioner's thigh biopsy combined with the normal result from her ankle biopsy suggests either a false negative or that the process is non-length dependent. *Id.* at 108-09.

Dr. Boylan discussed the sweat gland nerve fiber density results performed on August 21, 2017. Tr. at 57-58 (citing Ex. 8 at 66). The biopsy of Petitioner's left thigh found no sweat glands to analyze, while the biopsy of her left foot found "[s]kin with significantly reduced Sweat Gland Nerve Fiber Density, consistent with small fiber neuropathy." Ex. 8 at 66. Dr. Boylan explained that the absence of sweat glands in the left thigh biopsy meant that the test could not be interpreted and had to be disregarded as a diagnostic tool. Tr. at 57-58. The biopsy of the left foot contained sweat glands which showed a significant reduction in the density of sweat gland nerve fibers, which Dr. Boylan opined was consistent with SFN. *Id*. at 58-59. Dr. Boylan described Petitioner's skin biopsies as confirming the diagnosis of SFN. *Id*. at 59.

Dr. Boylan testified that Petitioner's feeling of difficulty standing, in the context of her previously reported dizziness, was "highly suspicious" of orthostatic hypotension. *Id*. at 62. This, in Dr. Boylan's view, supported an SFN diagnosis. *Id*. She explained that small nerve fibers are involved in the autonomic nervous system and that dizziness can be a manifestation of dysfunction in the autonomic nervous system. *Id*. Dr. Boylan stated that she commonly sees complaints of dizziness and light-headedness in patients with SFN in her clinical practice. *Id*. at 66-67. Additionally, the record notes that Petitioner's gait was occasionally unsteady, which Dr. Boylan opined could result from dizziness and is consistent with SFN. *Id.* at 67.

Dr. Boylan noted that Petitioner was experiencing allodynia[17] on January 24, 2018. Tr. at 75 (citing Ex. 57 at 1). She opined that allodynia is characteristic of SFN because small nerve fibers are involved in pain sensitivity and can regenerate abnormally. Tr. at 75-76. Dr. Buczyner also reported that "[t]emperature makes [Petitioner's] symptoms worse." Ex. 57 at 1. Dr. Boylan interpreted Dr. Buczyner's notes as stating that heat exacerbated Petitioner's allodynia and opined that this was also consistent with autonomic nervous system dysfunction. Tr. at 76-77.

Dr. Boylan testified that Dr. Buczyner's examination found "[d]ecreased pinprick in the legs in no particular pattern" and normal reflexes. Tr. at 77 (citing Ex. 57 at 2). Dr. Boylan opined that this was an abnormal result for a sensory examination and meant Petitioner had decreased sensation in her legs, but the sensory loss did not follow the "glove and stocking" distribution pattern. Tr. at 77-78. She opined that the glove and stocking distribution describes loss of sensation in a length dependent fashion. *Id*. Dr. Boylan testified that the absence of a pattern in Petitioner's decreased pin prick sensation is consistent with non-length dependent SFN. *Id*. She also testified that Petitioner's abnormal sensory examination with normal reflexes was consistent with SFN. *Id.*

Dr. Boylan opined that the five days from Petitioner's vaccination to the onset of her symptoms is an appropriate timeframe for vaccine-induced neuropathy. Tr. at 81-82. She added that when attributing neurologic syndromes to autoimmune etiologies, she prefers to see a delay of days to weeks between the antigen exposure and onset of the condition. *Id*. at 82. She also noted that it is easier to make the association with an antigenic exposure when the delay is measured in days because the proximity allows less time for potential intervening events. *Id*. Dr. Boylan concluded that five days post-vaccination is an appropriate time for autoimmune manifestation to begin. *Id*. at 83.

Dr. Boylan testified that it is appropriate to extrapolate the Table period of 3–42 days for onset of vaccine-induced GBS symptoms to SFN. *Id*. at 84. Dr. Boylan also stated that the mechanism which causes symptoms to begin from the triggering event is fundamentally the same between GBS and SFN. *Id*.

Dr. Boylan reiterated her opinion that the flu vaccination on September 29, 2014, caused Petitioner to develop acute isolated SFN. Tr. at 84. She explained that the acute onset, non-length dependency, and autonomic symptoms all favor immune-mediated SFN which she attributes to the flu vaccine, in part, because onset occurred five days after vaccination. *Id*. at 85-86. Dr. Boylan added that SFN is a very rare autoimmune-mediated condition and that she saw no evidence of diabetes, an antecedent infection, or any other explanation for Petitioner's condition in the medical records. *Id*. at 86-87. Dr. Boylan concluded that, within a reasonable degree of medical certainty, it was more likely than not Petitioner would not have developed SFN but for the flu vaccination. *Id*. at 87.

[17] Allodynia is "pain resulting from a non-noxious stimulus to normal skin." DORLAND'S, https:// www.dorlandsonline.com /dorland /definition?id=1820 (last visited May 25, 2023).

B. Respondent's Expert: Christopher Gibbons, MD, MMSc, FAAN, FAAS

1. Qualifications

Dr. Gibbons received his medical degree with distinction in immunology from Albert Einstein College of Medicine and a Master of Medical Science from a joint Harvard and MIT program. Ex. B ("Gibbons CV") at 1. He is board certified in neurology, clinical neurophysiology, and autonomic disorders. *Id*. at 20. Currently, Dr. Gibbons is an associate professor of neurology at Harvard Medical School and an attending physician in neurology at the Beth Israel Deaconess Medical Center and Joslin Diabetes Center. *Id.* at 1-2. Dr. Gibbons treats patients and educates physicians, residents, and medical students on neuropathy and small fiber neuropathy. Tr. at 123-25. Additionally, at the Beth Israel Deaconess Medical Center, Dr. Gibbons is the director of the neurocutaneous laboratory and associate director of the autonomic and peripheral nerve laboratory, as well as the director of the Joslin Diabetes Center neuropathy clinic. Gibbons CV at 2. He has published 61 peer-reviewed articles and 15 book chapters. *Id*. at 23-28. Dr. Gibbons' clinical work and research focus on autonomic and peripheral neuropathy. *Id*. at 30-31. I recognized him as an expert in the field of neurology and small fiber neuropathy. Tr. at 132.

2. Expert Reports

Dr. Gibbons submitted two expert reports in this case.

a. First Expert Report

In his first expert report, Dr. Gibbons opined that Petitioner did not have SFN and further, that her condition was not caused by the flu vaccine. Ex. A ("First Gibbons Rep."). Dr. Gibbons noted that Petitioner's reported symptoms of burning and tingling in her arms and feet could have been the result of a neuropathy, although he opined that it is unusual for a neuropathy to begin in the arms and then travel to the legs. First Gibbons Rep. at 3. He stated that "a length-dependent neuropathy starts in the legs, and moves proximally (towards the torso) to about the knee level, then it begins to affect the fingertips, then the hands." *Id.* Although Dr. Gibbons noted that acute non-length dependent neuropathies can occur, he stated that this has been rarely reported in the medical literature. *Id.* (citing Yuki, et al., *Acute painful autoimmune neuropathy: A variant of Guillain-Barré syndrome*, 57 MUSCLE & NERVE 320-24 (2018) (filed as Ex. A, Tab 2) ("Yuki"); Uncini & Yuki; Seneviratne & Gunasekera). He further noted that in all cases reported in the literature, patients exhibited sensory deficits on examination, but opined that there were no sensory deficits noted in Petitioner's case. *Id.*

Dr. Gibbons stated that Petitioner underwent repeated neurological examinations and EMG/NCS studies, all of which were normal. First Gibbons Rep. at 3. He noted that SFN can be difficult to detect but opined that Petitioner "saw several neurologists who documented normal sensory (including pinprick) examinations, which should not be normal in someone with a small fiber neuropathy." *Id.* Although one doctor noted distal sensory loss in the feet, these findings did not correlate with the skin biopsy findings "which were normal distally and abnormal at the more proximal site." *Id.* He opined that this further casts doubt on the skin biopsy findings. *Id.*

Dr. Gibbons stated that Petitioner's reports of weakness while standing and walking were not caused by SFN. First Gibbons Rep. at 4. He stated that SFN can cause burning, tingling, and shooting pains, but "small nerve fibers do not regulate strength, coordination, or walking." *Id.* Based on this, Dr. Gibbons would not conclude that Petitioner has SFN. *Id.*

Dr. Gibbons discussed skin biopsy testing and described it as "a standard tool for assessment of small fiber neuropathy." First Gibbons Rep. at 4. Biopsies are taken from standard locations at the distal leg, the distal thigh, and the proximal thigh to assess whether a patient has SFN. *Id.* He noted that the results of a skin biopsy must be evaluated in the context of a specific clinical case. *Id.* In this case, Petitioner reported pain in the left distal leg, yet she had a normal skin biopsy from this location. *Id.* Dr. Gibbons stated that Petitioner had a "a slightly reduced nerve fiber density count at the left distal thigh site" but indicated it was unclear whether Petitioner had ever reported pain in the legs above the knee. *Id.*

Dr. Gibbons also discussed that Petitioner had a sweat gland nerve fiber density test performed with these same biopsies. First Gibbons Rep. at 4. The results from the left distal leg were abnormal. *Id.* With respect to this result, Dr. Gibbons opined that Petitioner "has no clinically relevant symptoms to suggest a severe sudomotor[18] neuropathy (i.e. severe loss of sweating that leads to severely dry cracked skin), suggesting that this test is unlikely to be valid." *Id.* He noted that sweat gland nerve fiber density testing was never validated for clinical use, and that these results are frequently misinterpreted. *Id.*

Dr. Gibbons opined that there was no evidence that Petitioner had GBS. First Gibbons Rep. at 5. He reiterated that none of the EMG/NCS studies suggested GBS. He noted that there was no lumbar puncture performed. Additionally, Dr. Gibbons stated that Petitioner was not treated for GBS, which further suggests that her treating physicians did not believe this to be her correct diagnosis. *Id.*

Dr. Gibbons concluded by opining: "The weight of evidence in this case does not support a diagnosis of small fiber neuropathy, does not support a diagnosis of Guillain Barre syndrome, nor does a diagnosis of small fiber neuropathy explain the symptoms in this case." *Id.* at 6.

b. Second Expert Report

In his second expert report, Dr. Gibbons discussed his current research on SFN. Ex. C (hereinafter "Second Gibbons Rep.") at 3. He cited to a study published in 2020, noting that in this report, only 50% of patients with a biopsy reported as 'abnormal' by an outside laboratory had neuropathy on review of the pathology, and there were many errors in clinical examination or interpretation of clinical data that resulted in only 5 of 30 patients actually being eligible for the study." Second Gibbons Rep. at 3 (citing Gibbons, et al., *Clinical and pathological errors contribute to small fiber neuropathy misdiagnosis*, 25 J. PERIPHERAL NERVOUS SYSTEM 1 (2020) (abstract filed as Ex. C, Tab 1)). He reiterated his opinion that SFN is easy to misdiagnose. *Id.*

Turning to this case, Dr. Gibbons noted that Petitioner experienced pain and tingling in her

[18] The sudomotor nerves are "the nerves that innervate the sweat glands." DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id=92158 (last visited June 12, 2023).

arms, and problems with dexterity; Petitioner then developed a constant burning pain in her feet and arthralgias in her knees; after that, her symptoms ascended up her legs and resulted in difficulty with walking. Second Gibbons Rep. at 3. He noted that "all of these individual pieces of the puzzle do look like they could be small fiber neuropathy, but they do not fit together to make a coherent picture." *Id.* He again noted that Petitioner's regions of sensory loss "did not match up with the abnormalities on the skin biopsy." *Id.* He further opined that loss of dexterity and leg weakness is not consistent with SFN. *Id.* Although Dr. Boylan explained Petitioner's difficulty walking as related to orthostatic intolerance, "[o]rthostatic intolerance in the setting of an autonomic neuropathy is caused by orthostatic hypotension" and there is not documentation that Petitioner has this condition. *Id.* at 4. Ultimately, Dr. Gibbons expressed "skepticism" over a SFN diagnosis in Petitioner's case. *Id.*

Dr. Gibbons next discussed the skin biopsy results. Second Gibbons Rep. at 4. Although he agreed with Dr. Boylan that normative values for skin biopsies do not include symptoms, he stated that failing to consider a patient's clinical picture constitutes "a basic failure to understand disease pathophysiology and statistical likelihood." *Id.* With respect to the findings in Petitioner's case, Dr. Gibbons opined that "we have an abnormal biopsy in an asymptomatic region, and a normal biopsy in a region that seemed by examination and history to possibly be consistent with what could have been a small fiber neuropathy." *Id.*

Dr. Gibbons opined that Petitioner's skin biopsy results revealing 6.24 nerve fibers per millimeter was, in his opinion, "slightly abnormal." Second Gibbons Rep. at 4. He based this opinion on his clinical experience in interpreting these results over the past 20 years. *Id.* He further stated that "[a]lthough these results are not irrelevant, they are a fairly weak piece of data upon which to base a complex theory of small fiber neuropathy." *Id.* at 5. He concluded that the data supporting a SFN diagnosis in Petitioner's case to be "very weak." *Id.*

Although Dr. Gibbons acknowledged that a small number of patients with GBS present with SFN, he stated that "[i]n all reported cases, there are sensory deficits on examination, and the skin biopsy nerve counts were zero (no nerve fibers detected)." Second Gibbons Rep. at 5. He further noted that these patients often present with elevated protein levels in CSF. *Id.* Dr. Gibbons also stated that many other isolated SFN cases report severe autonomic dysfunction, which was not present in this case. *Id.* He agreed with Dr. Boylan that difficulty walking can occur in the context of orthostatic hypotension, it would present along with "postural lightheadedness, dizziness, syncope, coat hanger headache, orthodeoxia or platypnea."[19] None of those symptoms were present in this case. *Id.*

Dr. Gibbons opined that "[t]here is no association in the literature noted between a flu vaccination and development of small fiber neuropathy." Second Gibbons Rep. at 7. He concluded that "[t]he weight of evidence in this case does not support a diagnosis of small fiber neuropathy, does not support a diagnosis of Guillain Barre syndrome, nor does a diagnosis of small fiber neuropathy explain the symptoms in this case." *Id.* at 8.

[19] Platypnea is breathlessness or shortness of breath that is "induced by assumption of the upright position and relieved by assumption of a recumbent position." DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id=39526 (last visited June 9, 2023).

3. Testimony

Dr. Gibbons began by describing the process of evaluating and diagnosing patients with SFN. Tr. at 126. He testified that he begins with a detailed medical history, followed by physical and neurological examinations with a particular focus on the peripheral nervous system. *Id.* The examination would include strength examinations, large fiber evaluations, vibration detection, temperature detection, and testing pinprick sensation. *Id.* at 127. Dr. Gibbons added that the totality of a patient's coordination, gait, and reflexes would also be considered. *Id.* He stated that those examinations are the standard workup for his patients. *Id.*

Dr. Gibbons testified that diagnosis of SFN involves a combination of history, examination findings, and ancillary testing such as skin biopsy. Tr. at 134. He stated that a sensory exam involves evaluating what a patient feels in response to a stimulus such as touch, temperature, vibration, movement, or pin prick. *Id.* at 135. He opined that loss of sensation (particularly to pin prick, temperature, vibration, or movement), allodynia, and hyperalgesia[20] are clinical findings that support a diagnosis of SFN. *Id.* at 136-37. He added that tests of reflexes and strength are also part of the clinical exam. *Id.* at 137.

Dr. Gibbons testified that EMG and NCS are diagnostic tools for large nerve fibers. Tr. at 138. NCS involves "measuring the transfer of electricity along the nerve," which indicates whether the myelin sheath is intact. *Id.* at 139. This in turn gives an "idea of the health of the peripheral nerve large fiber system." *Id.* EMG, on the other hand, evaluates the health of large nerve fibers that lead into muscles and control muscle function. *Id.* Dr. Gibbons opined that EMG and NCS are not diagnostic of SFN, but that they can rule out large fiber involvement. *Id.*

Dr. Gibbons explained that a skin biopsy involves obtaining a small skin sample and staining it such that the nerves are visible and comparing the number of nerve fibers in the sample to normative values based on data from large populations of people. Tr. at 140. He testified that skin biopsy is widely used to confirm or rule out a diagnosis of SFN. *Id.* He opined that skin biopsy is a "reasonably good" diagnostic tool in terms of sensitivity and specificity, but that it is not definitive. *Id.* He noted that different physicians may have slightly different techniques for taking a biopsy sample and that different commercial laboratories may perform the test in slightly different ways, resulting in an 80 to 90 percent degree of specificity. *Id.* at 141.

Dr. Gibbons testified that there are a number of causes of the isolated form of SFN, including metabolic disorders such as diabetes, vitamin deficiencies, malignancies, certain medications such as chemotherapy, acute neuropathies such as GBS, and autoimmune disorders. Tr. at 142-43. He stated that approximately 30 percent of cases are idiopathic. *Id.* at 143.

Dr. Gibbons testified that, in his experience, he has never seen a case where the flu vaccine caused SFN and that he is not aware of medical literature describing such a case. Tr. at 145-46.

He opined that the term "orthostatic intolerance" means that the patient feels unwell when she stands up and that it can have several causes. Tr. at 146. He added that orthostatic hypotension

[20] Hyperalgesia is "abnormally increased nociception (pain sense)." DORLAND'S, https:// www.dorlandsonline.com /dorland/definition?id=23666 (last visited June 27, 2023).

is a decrease in blood pressure that occurs upon standing and is a specific diagnosis that Petitioner's records do not document. *Id.* at 146-47. Dr. Gibbons testified that Petitioner's dizziness upon standing, trouble walking, and lack of coordination were not indicative of an autonomic neuropathy. *Id.* at 146. He opined that an autonomic neuropathy alone would not be enough to cause orthostatic hypotension of sufficient magnitude to cause symptoms. *Id.* at 147. He further opined that a typical early sign of autonomic neuropathy is resting tachycardia, or an increase in heartrate while seated. *Id.* He testified that Petitioner's medical record does not show evidence of resting tachycardia and that Petitioner reported no constipation, diarrhea, bladder dysfunction, or problems with sweating. *Id.*

Dr. Gibbons testified that, based on the entire medical record, he was not convinced that Petitioner has SFN. Tr. at 148. He agreed that multiple treating physicians included SFN in their differential diagnoses but opined that he did not see any evidence that SFN was the correct diagnosis. *Id.* He noted that multiple examinations documented Petitioner's deficits but stated that the deficits were different in each exam. *Id.* at 149. He testified that the medical record was inconsistent and that Petitioner's skin biopsy results did not match Petitioner's description of her symptoms. *Id.* He opined that because of the "disparate pathology, history, [and] exam," he was unable to diagnose Petitioner with SFN. *Id.* Dr. Gibbons conceded that certain aspects of Petitioner's clinical presentation suggested that SFN belonged in the differential diagnosis, but he maintained that the overall picture did not support SFN. *Id.* at 149-50. He testified that while the skin biopsy results were abnormal, the samples were taken from areas that Petitioner did not indicate were symptomatic. *Id.* at 150. He opined that the abnormal biopsy results were a false positive and had no diagnostic value. *Id.*

Dr. Gibbons testified that there is no evidence Petitioner had GBS. Tr. at 152. He opined that GBS sometimes results in small fiber damage, but that this is due to a bystander effect rather than a direct attack (i.e., small fibers sustain damage from the body's inflammatory response because they are in close proximity to large fibers). *Id.* Dr. Gibbons opined that a small number of cases have been reported that suggest that there is an isolated small fiber neuropathy in GBS but that all such cases have also involved elevated protein in cerebrospinal fluid ("CSF"). *Id.* at 152-53. He stated that GBS is a treatable condition, and that Petitioner was never treated for it. *Id.* at 153. He also noted that Petitioner never underwent a lumbar puncture. *Id.*

Dr. Gibbons disagreed with Dr. Boylan's testimony that GBS can manifest solely as SFN. Tr. at 156. He opined that there may be some overlap between the two, such as when large fiber pathology affects small fibers, but did not believe that this was a true isolated SFN. *Id.* He testified that he has never seen GBS manifest as SFN in his clinical practice. *Id.*

Dr. Gibbons cited medical literature in support of his opinion in this case. Tr. at 156-60. He noted that Yuki reported three patients with autonomic symptoms, skin biopsies showing no nerves in the complained-of area, absent or decreased reflexes, and elevated CSF protein, indicating both large and small fiber involvement. Tr. at 157. He opined that Petitioner's reflexes were normal throughout and that we cannot know whether her CSF protein was elevated because she never underwent a lumbar puncture. *Id.* at 158. Dr. Gibbons further testified that Yuki suggests that there is not an isolated small fiber neuropathy, but that large fibers are also involved. *Id.* Dr. Gibbons also noted that Uncini & Yuki reported six cases with varying presentations and proposed

a typology of GBS variants. *Id.* at 159 (citing Uncini & Yuki, *Sensory Guillain-Barré syndrome and related disorders: An attempt at systematization*, 45 MUSCLE & NERVE 464-70 (2012) (filed as Ex. A, Tab 3) ("Uncini & Yuki")). Finally, Dr. Gibbons noted that Seneviratne & Gunasekera also reported six cases in which all patients had elevated CSF protein and recovered fully within a few months. Tr. at 160-61. He opined that this is inconsistent with Petitioner's reports that her symptoms still persisted. *Id.* at 161.

Dr. Gibbons opined that he was unaware of any medical literature suggesting that the flu vaccine can cause SFN. Tr. at 161. He stated that there is some evidence that GBS can cause SFN. *Id.* at 162. He further stated that he encourages his patients to get the flu vaccine and opined that the flu vaccine did not cause Petitioner to develop any kind of neuropathy. *Id.*

Dr. Gibbons opined that Petitioner's three treating neurologists and Dr. Sharma all made different findings on exam. Tr. at 184. Based on this, he concluded that there was some difficulty in coming to a clear diagnosis in Petitioner's case and reiterated his opinion that SFN is not the correct diagnosis. *Id.* at 184-85.

Dr. Gibbons testified that it is challenging to determine the cause of an acute neuropathy. Tr. at 196. He opined that up to two months was an appropriate timeframe for a vaccine to trigger a neuropathy, and that, with a vaccine administered annually like the flu vaccine, there is a one in six chance of onset of neuropathy occurring post-vaccination by coincidence. *Id.*

Dr. Gibbons testified that it is not impossible that an infection might cause SFN by the same mechanism by which the flu vaccine can cause GBS, but he stressed that nerve damage in GBS results from inflammation rather than direct attack on nerve fibers. Tr. at 219. He agreed that molecular mimicry is generally accepted as a mechanism by which the flu vaccine can cause GBS. *Id.* at 221.

Dr. Gibbons conceded that Petitioner's symptoms may be consistent with SFN and that Petitioner's treating physicians diagnosed her with SFN. Tr. at 236. He agreed that Petitioner's skin biopsy, the "gold standard" for diagnosing SFN, was abnormal. *Id.* at 237. He opined that while Petitioner's symptoms "sound like [SFN]," he saw no evidence of an immune reaction in the medical record. *Id.* at 242.

VI. Applicable Law

A. Petitioner's Burden

Under the Vaccine Act, a petitioner may prevail in one of two ways. First, a petitioner may demonstrate that she suffered a "Table" injury—i.e., an injury listed on the Vaccine Injury Table that occurred within the time period provided in the Table. § 11(c)(1)(C)(i). "In such a case, causation is presumed." *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed in the Vaccine Injury Table, a petitioner may demonstrate that she suffered an "off-Table" injury. § 11(c)(1)(C)(ii).

For both Table and non-Table claims, Vaccine Program petitioners bear a "preponderance

of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1324 (Fed. Cir. 2010); *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873* (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records of by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Secretary of Health and Human Services*. 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires that petitioner establish by preponderant evidence that the vaccinations he received caused her injury "by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* at 1278.

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs*., 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325-26). Special Masters, despite their expertise, are not empowered by statute to conclusively resolve what are complex scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Hum. Servs*., 121 Fed. Cl. 230, 245 (2015) ("[p]lausibility … in many cases may be enough to satisfy *Althen* prong one" (emphasis in original)), *vacated on other grounds*, 844 F.3d 1363 (Fed. Cir. 2017). But this does not negate or reduce a petitioner's ultimate burden to establish

her overall entitlement to damages by preponderant evIdence. *W.C. v. Sec'y of Health & Hum. Servs*., 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, because they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing … that mandates that the testimony of a treating physician is sacrosanct–- that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence also present in the record–- including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs*., 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Health & Hum. Servs*., No. 06-522V 2011 WL 1935813 at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 Fed. App'x 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs*., 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs*., 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand on other grounds*, 105 Fed. Cl. 353 (2012), *aff'd without op.,* 503 F. App'x 952 (Fed. Cir. 2013). *Koehn v. Sec'y of Health & Hum. Servs*., No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. Law Governing Analysis of Fact Evidence

The process for making factual determinations in Vaccine Program cases begins with analyzing the medical records, which are required to be filed with the petition. Section 11(c)(2). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs*., 3 F.3d 413, 417 (Fed. Cir. 1993) (it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

Medical records created contemporaneously with the events they describe are generally trustworthy because they "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions," where "accuracy has an extra premium." *Kirby v. Sec'y of Health & Hum. Servs.*, 997 F.3d 1378 (Fed. Cir. 2021) citing *Cucuras*, 993 F.2d at 1528. This presumption is based on the linked proposition that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11-685V, 2013 WL 1880825 at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) *mot. for rev. denied*, 142 Fed. Cl. 247, 251-52 (2019), *vacated on other grounds and remanded*, 809 Fed. Appx. 843 (Fed. Cir. Apr. 7, 2020).

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs.*, No. 03-1585V, 2005 WL 6117475 at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony— especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; see also *Murphy v. Sec'y of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. U.S. Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, there are situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs.*, 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475 at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should

be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent and compelling." *Sanchez*, 2013 WL 1880825 at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs.*, No. 90-2808V, 1998 WL 408611 at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *LaLonde v. Sec'y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203-04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. Analysis of Expert Testimony

Establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of his or her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594-96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (*citing Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). "The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community." *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592-95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora. *Daubert* factors are employed by judges to exclude evidence that is unreliable and potentially confusing to a jury. In Vaccine Program cases, these factors are used in the weighing of the reliability of scientific evidence. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66-67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate persuasiveness and reliability of expert testimony has routinely been upheld. *See*, e.g., *Snyder*, 88 Fed. Cl. at 743. In this matter, (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner's

case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)). A "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly*, 592 F.3d at 1324. Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Id.* at 1325-26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. Consideration of Medical Literature

Finally, although this decision discusses some but not all of the medical literature in detail, I have reviewed and considered all of the medical records and literature submitted in this matter. *See Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); *Simanski v. Sec'y of Health & Hum. Servs.*, 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'" (citation omitted)), *aff'd*, 601 F. App'x 982 (Fed. Cir. 2015).

VII. Analysis

In the context of the Program, "to establish causation, the standard of proof is preponderance of evidence, not scientific certainty." *Langland v. Sec'y of Health & Hum. Servs.*, 109 Fed. Cl. 421, 441 (2013). Petitioner's burden under *Althen*'s first prong is to provide a medical theory causally connecting the vaccination and the injury. *Id.* This theory must be sound and reliable. *Boatmon*, 941 F.3d at 1359. For the reasons discussed in detail below, I find that Petitioner has provided a sound and reliable medical theory causally connecting her flu vaccination to SFN.

A. Petitioner's Diagnosis

As a threshold matter, a petitioner must establish that she suffers from the condition for which she seeks compensation. *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1346 (Fed. Cir. 2010). "The function of a special master is not to 'diagnose' vaccine-related injuries, but instead to determine 'based on the record as a whole and the totality of the case, whether it has been shown by a preponderance of the evidence that a vaccine caused the [petitioner]'s injury.'" *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1382 (Fed. Cir. 2009) (quoting *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 549 (Fed. Cir. 1994)). "Although the Vaccine Act does not require absolute precision, it does require the petitioner to

establish an injury -- the Act specifically creates a claim for compensation for 'vaccine-related injury or death.'" *Stillwell v. Sec'y of Health & Hum. Servs.*, 118 Fed. Cl. 47, 56 (2014) (quoting 42.U.S.C. § 300aa-11(c)). Accordingly, the Federal Circuit has established that it is "appropriate for the special master to first determine what injury, if any, [is] supported by the evidence presented in the record" before applying a causation analysis pursuant to *Althen v. Secretary of Health & Hum. Services,* 418 F.3d 1274 (Fed. Cir. 2005). *Lombardi*, 656 F.3d at 1351-53. In this case, Petitioner has preponderantly demonstrated that she suffers from SFN.

Although Drs. Boylan and Gibbons disagree as to Petitioner's diagnosis, they are in agreement that, in general, the diagnosis of SFN involves analysis of the patient's medical history and exam findings with the possibility of objective testing to confirm the diagnosis (e.g., skin biopsy or imaging). Tr. at 37, 134. I will analyze the evidence pertaining to each component in turn.

1. Medical History

At the entitlement hearing, Dr. Boylan described the typical clinical presentation of SFN as burning pain, itching, and electrical sensations, as well as hypersensitivity to pain, changes in sweating, and skin discoloration. Tr. at 60, 75-76. She also opined that dizziness and lightheadedness are common in SFN. *Id.* at 66-67. She testified that Petitioner's medical history was consistent with SFN. *Id.* at 63. Dr. Gibbons agreed that burning and tingling in the extremities, as well as allodynia and hyperalgesia, support a diagnosis of SFN. First Gibbons Rep. at 3; Tr. at 136-37. He added that loss of sensation also supports a diagnosis of SFN. Tr. at 136-37. However, Dr. Gibbons concluded that weakness in the legs and orthostatic intolerance in Petitioner's medical history was not consistent with SFN. Second Gibbons Rep. at 3-4. Ultimately, Dr. Gibbons opined that "[t]he challenge in this case is all of these individual pieces of the puzzle do look like they could be small fiber neuropathy, but they do not fit together to make a coherent picture." *Id.* at 3.

I find that Petitioner's medical history supports her claim that she had SFN. As soon as her visit to Dr. Urban on November 12, 2014, Petitioner's neurological symptoms are well-documented, beginning with tingling. Ex. 7 at 40. This was closely followed by reports of burning pain in her feet, tingling in her hands and feet, and decreased pinprick sensation on November 20. Ex. 4 at 9. Petitioner's reports of burning pain are consistent throughout the medical record. *See, e.g.,* Ex. 2 at 7; Ex. 6 at 1; Ex. 10 at 5-6; Ex. 13 at 11. Tingling is also documented consistently in Petitioner's medical records. *See, e.g.,* Ex. 4 at 9; Ex. 2 at 7; Ex. 8 at 19; Ex. 3 at 13-15. Petitioner additionally experienced allodynia, which Dr. Boylan described as characteristic of SFN. *See, e.g.*, Ex. 8 at 12 (August 17, 2017 medical appointment noting that Petitioner was bothered by things touching her legs); Ex. 57 at 1 (January 24, 2018 appointment documenting that Petitioner "does not like having anything touch her skin"); Tr. at 76.

In addition, Dr. Boylan testified that there is no evidence in Petitioner's medical history of an alternative cause for her symptoms, such as diabetes or infection. Tr. at 86-87. Dr. Gibbons expressed uncertainty as to what he believes the correct diagnosis is in Petitioner's case, but he suggested fibromyalgia. Tr. at 246.

In total, Petitioner's medical history supports her position that she has SFN.

2. Exam Findings

Both experts emphasized the importance of abnormal findings on neurological exam to a diagnosis of SFN. Dr. Gibbons testified that the standard examination in a suspected case of SFN would involve strength examinations, large fiber evaluations, vibration detection, and testing pinprick sensation. Tr. at 127. He added that the totality of a patient's coordination, gait, and reflexes would also be considered. *Id.* Dr. Gibbons stated that loss of pin prick sensation, loss of temperature sensation, allodynia, and hyperalgesia on clinical exam support a diagnosis of SFN. *Id.* at 136. He testified that strength examination, reflexes, vibration, and proprioception[21] are "standard large fiber modalities." *Id.* at 137. Dr. Gibbons opined that examination involving light touch could apply to both large and small fiber conditions. *Id.* Dr. Boylan agreed that pinprick testing is part of the examination for SFN, but also cited literature finding that pinprick sensation was normal in 30% of patients with SFN in the study. Second Boylan Rep. at 3 (citing Blackmore & Siddiqi). She also stated that sensation of temperature and vibration and joint position sense would also be considered. Tr. at 40. Finally, Dr. Boylan testified that examination of the patient's limbs for trophic changes, discoloration, and swelling is also part of the diagnosis of SFN. *Id.*

In his first expert report, Dr. Gibbons opined that Petitioner's medical record did not contain evidence of sensory deficits. First Gibbons Rep. at 3. However, he testified at the hearing that Petitioner's decreased pin prick sensation documented in her medical records is, in fact, a sensory deficit. Tr. at 211. Dr. Gibbons pointed to the differences in clinical exams to support his position that Petitioner did not have SFN. He testified: "the challenge I'm having in this case is I see multiple exams that show deficits, but the deficits are different in every exam and then … exams where there's entirely no deficits … interspersed throughout the record." *Id.* at 149.

Petitioner's medical records document abnormal small fiber findings at multiple points during the course of her condition. Dr. Alboukrek noted Petitioner exhibited decreased pinprick sensation in her feet on November 20, 2014, and on December 18, 2014. Ex. 4 at 8, 10. On January 13, 2015, Dr. Sharma's sensory examination revealed impaired light touch and pinprick sensation in the median nerve distribution in both of Petitioner's hands and distally in her feet. Ex. 6 at 3. On August 13, 2015, Dr. Schoenhaus' examination found that Tinel sign was present on the left and right posterior tibial nerves and the left and right superficial peroneal nerve. Ex. 10 at 6. On January 24, 2018, Dr. Buczyner's sensory examination found decreased pinprick in the legs in no particular pattern. Ex. 57 at 2. Dr. Boylan testified this finding is consistent with a non-length dependent small fiber neuropathy. Tr. at 78.

Although the examinations conducted by Petitioner's various medical providers are not all consistent, a substantial number of them document that Petitioner was experiencing sensory deficits and other indications of SFN. I find that the evidence of abnormal sensory examinations support Petitioner's contention that she has SFN.

[21] Proprioception: "[T]he reception of stimuli produced within the organism." www.merriam-webster.com/dictionary/the%20reception%20of%20stimuli%20produced%20within%20the%20organism (last accessed Oct. 19, 2023).

3. Objective Testing

Petitioner underwent a skin biopsy of her left thigh and left calf/foot on August 21, 2017, that assessed epidermal nerve fiber density and sweat gland nerve fiber density. Ex. 11 at 8. The results of her left thigh biopsy showed "significantly reduced Epidermal Nerve Fiber Density, consistent with small fiber neuropathy," but no sweat glands could be identified for analysis. Ex. 8 at 63, 66. The results of her left calf/foot biopsy showed normal epidermal nerve fiber density and "significantly reduced Sweat Gland Nerve Fiber Density, consistent with small fiber neuropathy." *Id.*

a. Epidermal Nerve Fiber Density

At the outset, I note that it is not entirely clear where the biopsy from Petitioner's lower leg was taken. There is some evidence that the sample was taken from either her foot or her calf. Tr. at 207. The procedure documentation section of the medical record describes the location as "calf." Ex. 8 at 60. The pathology report documenting the results of the test indicates the sample was taken from the foot. *Id.* at 63. Petitioner testified at the entitlement hearing that her lower leg biopsy was taken between her ankle and her calf, closer to the calf but above the ankle. Tr. at 254-55. As this testimony is consistent with the medical record which documented the biopsy site, I find it to be persuasive. I conclude that preponderant evidence supports the biopsy was taken from Petitioner's lower calf.

The parties' experts agreed that the results of skin biopsy are often a component of the analysis leading to a diagnosis of SFN. Tr. at 48-49, 134. Both parties filed medical literature supporting this contention. Smith & Gibson, *Skin biopsy for the evaluation of peripheral nerve disease*, UP TO DATE (2020) ("Smith & Gibson") (filed as Ex. 50) ("Skin biopsy can be used for the diagnosis of any distal symmetric polyneuropathy including those due to the involvement of small unmyelinated or large unmyelinated fibers."); Gibbons, Christopher H., *Small Fiber Neuropathies*, 20(5) CONTINUUM 1398-1412, 1402 (2014) ("Gibbons") (filed as Ex. A-1) ("Skin biopsy has become the pathologic gold standard used for the diagnosis of a small fiber neuropathy."). Dr. Gibbons agreed that skin biopsy testing has a greater than 90% specificity. Second Gibbons Rep. at 4. Additionally, there was a 5% chance that the study would be abnormal "based on the 5th percentile cut-off normative value." *Id.* at 5.

Dr. Boylan opined that Petitioner's skin biopsy results support a "definitive" diagnosis of SFN. Second Boylan Rep. at 5. Dr. Gibbons opined that Petitioner's skin biopsy results were "slightly abnormal," and that while not irrelevant, this was a "fairly weak" piece of data on which to base a diagnosis of SFN. Second Gibbons Rep. at 4-5. Dr. Gibbons emphasized that Petitioner's reported symptoms were dislocated on her body from the site of her abnormal biopsy results and that the biopsy thus had no diagnostic value. Tr. at 150. He opined that the skin biopsy has greater value to the diagnosis if the complaints are in the same location as the abnormal biopsy result. *Id.* at 207. In particular, he noted that Petitioner reported a burning sensation in her feet, but the epidermal nerve fiber density in her left foot was normal. *Id.* at 245. As discussed above, however, it is more likely that the biopsy was taken from Petitioner's lower calf.

I find that the results of Petitioner's skin biopsy support a diagnosis of SFN. The pathologist who prepared the report indicated that the Petitioner's significantly reduced epidermal nerve fiber density results were consistent with small fiber neuropathy. Ex. 8 at 63. In light of the expert testimony, I find this result to be particularly significant. I note Dr. Gibbons' opinion regarding the dislocation of Petitioner's symptoms and her abnormal biopsy results is not consistent with Petitioner's testimony or the medical records.

Petitioner complained of burning pain and weakness in her legs that began in November 2014. Ex. 6 at 1. In February 2015, Dr. Fernyhough noted that Petitioner had "enigmatic post vaccine neuropathy of the feet and distal legs distribution." Ex. 8 at 21. Petitioner reported leg pain to Dr. Davenport on June 4, 2015. Ex. 3 at 13-15. Further, Petitioner testified that she experienced sensations of pain, burning, and tingling in both legs, over the length of the entire leg, to include the thigh. Tr. at 254-55. This testimony is consistent with the medical records, and suggests that Petitioner's symptoms correlate with the skin biopsy results, which Dr. Gibbons has agreed is the "gold standard" in diagnosing small fiber neuropathy. Tr. at 221, 224, 237.

Although Petitioner's lower leg biopsy was normal with regard to epidermal nerve density, Dr. Boylan opined that this did not impact her diagnosis because an abnormal result is highly likely to be accurate while a normal result is more likely to be a false negative. Tr. at 55-56. Petitioner filed medical literature that supports both the importance of the analysis of epidermal nerve fiber density as a diagnostic tool for SFN and the analytical limitations of skin biopsy as a testing methodology. Hays, Arthur P., *Utility of Skin Biopsy to Evaluate Peripheral Neuropathy*, 10 CURRENT NEUROLOGY & NEUROSCIENCE REPS. 101-07, 104 (2010) ("Hays") (filed as Ex. 49) ("[A] negative result does not exclude [small fiber sensory neuropathy]. In view of the inherent limitations of skin biopsy, the results must be interpreted carefully together with clinical findings and other paraclinical tests.").

Accordingly, the weight of the evidence regarding Petitioner's epidermal nerve fiber density skin biopsy results supports her contention that she has SFN.

b. Sweat Gland Nerve Fiber Density

Dr. Boylan opined that the sweat gland nerve fiber density (SGNFD) result in Petitioner's thigh was not useful as a diagnostic tool because the test found no sweat gland nerve fibers to analyze. Tr. at 57-58. She commented that, as a general matter, sweat gland nerve fiber density is less useful diagnostically because it is less standardized than intraepidermal nerve fiber density. *Id.* at 50, 58. Dr. Gibbons opined that the sweat gland test performed in this case had no clinical significance. Tr. at 232. He testified that "six times out of ten it will be wrong, just by chance." Tr. at 233. He provided several bases for this opinion.

Dr. Gibbons indicated that the technique for testing sweat glands was based on his research and noted that the report cites to this research. Tr. at 186; Ex. 8 at 65. He further testified that the test is not viable when assessing individual patients. Tr. at 187.

> So in a single patient, we never found that that test was viable. What you could do is compare two groups and with enough volume and enough data you could see

> differences between groups. And that, in fact, was our publication, which they use, and they actually use our normative values, but the problem is this was never a normative value for a single patient. Because on any single biopsy, you may get one biopsy -- sweat gland, you may get none, you may get 50.

Tr. at 187. Dr. Gibbons also persuasively discussed the results of the SGNFD result in Petitioner's case. The abnormal result in her lower extremity did not correlate with the normal epidermal nerve fiber density in that same location. According to Dr. Gibbons, it is extremely rare to only have an abnormal SGNFD in the context of a normal epidermal nerve fiber density result. Tr. at 188.

Ultimately, I find Dr. Gibbons' opinion to be persuasive on this issue. Accordingly, I have not considered Petitioner's abnormal SGNFD result in my analysis of whether the weight of the evidence supports SFN as her correct diagnosis.

4. Treating Physicians

When weighing evidence, special masters should consider the opinions of treating physicians. *Capizzano*, 440 F.3d at 1326. The opinions of treating physicians about the appropriate diagnosis are often persuasive because the physicians have direct experience with the patient whom they are diagnosing. *See McCulloch v. Sec'y of Health & Hum. Servs.*, No. 09-293V, 2015 WL 3640610, at *20 (Fed. Cl. Spec. Mstr. May 22, 2015).

It is clear from the medical record that Petitioner's treating physicians did not arrive at a diagnosis of SFN immediately. *E.g.,* Ex. 8 at 5 (Dr. Ukani's assessment on November 4, 2014, was myalgia and chronic SSRI use); Ex. 2 at 8 (Dr. Bailyn's impression on December 4, 2014, was limb dysesthesia with no indication of generalized peripheral nerve dysfunction). However, later medical records contain repeated and consistent references to SFN. On January 13, 2015, Dr. Sharma assessed Petitioner's symptoms as suggesting small fiber dysfunction. Ex. 6 at 3. On June 4, 2015, Dr. Davenport assessed her with bilateral lower extremity numbness consistent with either mild GBS or small fiber neuropathy. Ex. 3 at 14. On August 17, 2017, Dr. Buczyner assessed Petitioner with neuropathy that may represent a small fiber neuropathy or CIDP. Ex. 8 at 12. After the skin biopsy results, Dr. Buczyner diagnosed Petitioner with SFN. Ex. 57 at 2.

Over a period of approximately two and a half years, several different providers concluded that SFN was a likely cause of Petitioner's symptoms. Accordingly, I conclude that the weight of the evidence of treating physician opinions supports Petitioner's claim that she had SFN.

Based on the above, I conclude that Petitioner's diagnosis of SFN is supported by a preponderance of the evidence.

B. Evidence that SFN is a Variant of GBS

I am not bound by the decisions of other special masters. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998), *aff'd* 191 F.3d 1344 (Fed. Cir. 1999). Prior cases can, however, have persuasive value. I note that I am not first among my colleagues to conclude that SFN is a variant of GBS. In *Swaiss v. Secretary of Health and Human Services*, a special master found that

the petitioner had "adequately supported the existence of an immune-mediated small fiber neuropathy, which may be referred to as a small fiber GBS variant." No. 15-286V, 2019 WL 6520791, at *13 (Fed. Cl. Spec. Mstr. Nov. 4, 2019). Similarly, in *Doe v. Secretary of Health and Human Services*, a special master found that the petitioner had preponderantly shown that the flu vaccine caused her serum sickness and SFN, accepting the petitioner's evidence that GBS and SFN were sufficiently similar to be analogous for purposes of her theory of causation. No. [redacted]V, 2007 WL 3120297, at *7-8 (Fed. Cl. Spec. Mstr. Oct. 18, 2007).

Dr. Boylan opined that Petitioner "has an atypical variant of GBS manifesting as an acute onset, auto-immune mediated and non-length dependent small fiber neuropathy. [Petitioner's] illness was caused by the influenza vaccination administered to her on September 29th, 2014." Second Boylan Rep. at 9. She cited medical literature supporting the contention that there are several variants of GBS that differ in terms of their clinical and electrodiagnostic presentations. *Id.* at 8; Seneviratne & Gunasekera at 540 ("[S]everal variants of [GBS] with different clinical manifestations have been reported."); Uncini & Yuki at 464 (recognizing reports in the literature of several types of GBS with different clinical presentations and electrophysiological findings).

Dr. Boylan opined that SFN should be considered a variant of GBS, and that instances of small fiber damage are not uncommon in cases of classic GBS. Tr. at 41. She added that sensory GBS is a demyelinating condition that is detectable with EMG, but that isolated SFN is not. *Id.* at 42-43. She opined that isolated SFN can be a presentation of GBS, but that such events are rare. Second Boylan Rep. at 6. Dr. Gibbons agreed that "[a]lthough exceedingly rare, isolated small fiber neuropathy can be a presentation of [GBS]." First Gibbons Rep. at 5.

Dr. Boylan opined that there is evidence in the literature that SFN as a manifestation of GBS, like classic GBS, is immune mediated. Second Boylan Rep. at 9. She cited a study by Levine in which blood serum testing of 155 patients with biopsy-confirmed SFN revealed that 48% of patients with SFN of unknown etiology also had two specific antibodies. Levine at 514. Levine concluded that "[t]hese data suggest that serum IgM binding to TS-HDS may be considered as a potential autoimmune association for SFN." *Id.* Levine further noted that the antibodies identified "were associated with subgroups of SFN, acute-onset, female, and non-length-dependent pathology." *Id.* Dr. Boylan opined that the similarities between the study population and Petitioner were "uncanny." Second Boylan Rep. at 9.

Dr. Gibbons opined that there is no evidence that Petitioner had GBS. In particular, he cited her normal EMG results and the lack of spinal tap to confirm or rule out elevated protein in her CSF. First Gibbons Rep. at 3, 5-6. Abnormal EMG and elevated levels of the protein albumin in CSF (referred to as "albuminocytologic dissociation") are two important objective indicators supporting a diagnosis of classic GBS. Vriesendorp at 2. However, Dr. Boylan and Dr. Gibbons testified that EMG detects damage to large nerve fibers (the target of demyelination in classic GBS) but cannot detect small fiber damage. Tr. at 37-38, 138-39; *see also*, Pan at 387 ("Conventional nerve conduction studies only detect abnormalities of large-diameter sensory nerves and offer no information regarding the degeneration of small-diameter sensory nerves."). Furthermore, the medical literature submitted provides support for the conclusion that elevated protein in CSF supports a GBS diagnosis but is not a strict requirement. Uncini & Yuki at 464 ("[A]lbuminocytologic dissociation…[was] considered helpful in individuating patients with

sensory GBS, but [was] not a prerequisite."); Vriesendorp at 6-7 (listing albuminocytologic dissociation as a "supportive feature" rather than a "required feature" for purposes of GBS diagnosis).

Petitioner cited medical literature in support of her position. First, Petitioner relied on Uncini & Yuki, in which the authors recognize that there have been a small number of cases reported in which patients diagnosed with acute sensory neuropathy may in fact have GBS. Uncini & Yuki at 464. They propose a classification system for different sub-types of sensory GBS "based on the prevalent size (large or small) of fibers involved and the primary site of damage (myelin, axon, and/or primary sensory neuron)." *Id.* at 469. In their literature review, Uncini & Yuki operatively define sensory GBS as "an acute, monophasic, widespread neuropathy characterized clinically by exclusive sensory symptoms and signs that reach their nadir in 6 weeks." *Id.* Elevated protein in CSF was considered helpful, but not necessary, in identifying patients with sensory GBS. *Id.* Uncini & Yuki identified ten patients whom they describe as having acute sensory small fiber neuropathy-ganglionopathy, with symptoms including (1) acute onset of numbness and/or burning and pain in extremities; and (2) distal sensory loss for pain and temperature. *Id.* at 467. These patients had normal nerve conduction studies, normal proprioception, and normal vibration sense. *Id.* Six of the seven patients who underwent spinal taps had elevated protein in their CSF. *Id.* The authors also suggest that five of the ten patients described "may represent cases of post-vaccination GBS involving small fibers," having been vaccinated for rabies, varicella, or Lyme disease one day to two months before symptom onset. *Id.* Dr. Boylan referred to Figure 1 in Uncini and Yuki's article during her testimony. Tr. at 46 (citing Uncini & Yuki at 468). The chart in Figure 1 identifies three different types of sensory GBS, with two types affecting large nerve fibers and the third, acute sensory small fiber neuropathy-ganglionopathy, affecting small fibers. Uncini & Yuki at 468. Dr. Boylan opined that this is the variant of GBS that Petitioner has. Tr. at 46.

Second, Petitioner relied on Seneviratne & Gunasekera, who state that "[t]he existence of sensory [GBS] has now been established beyond doubt and it is shown to be a demyelinating neuropathy electrophysiologically." Seneviratne & Gunasekera at 540. In their report on six patients, the authors note that all six had elevated protein in CSF, normal nerve conduction studies, normal strength, and normal reflexes. *Id.* at 540-41. They note that these six patients meet seven of the nine diagnostic criteria for GBS and demonstrate clinical features compatible with SFN. *Id.* at 541-42. They posit that these six patients may represent a subtype of sensory GBS that they refer to as acute small fiber sensory neuropathy. *Id.* at 542.

Third, Petitioner cited Yuki, which evaluated three patients who presented with severe pain in their extremities, impaired pinprick sensation, and allodynia some weeks after infectious illness. Yuki at 320. The authors contend that these three patients, along with six similar patients reported in previous case studies, "suggest that an acute autoimmune response can be directed against small fibers and exhibit similarities to [GBS]." *Id.* at 323.

Petitioner filed Pan et al., *Cutaneous innervation in Guillain-Barré syndrome: pathology and clinical correlations*, 126 BRAIN 386-97 (2003) (filed as Ex. 20) ("Pan"). The authors in Pan found diminished epidermal innervation in some GBS patients, suggesting that "small-fiber sensory neuropathy is also an important manifestation of GBS, and that GBS should be considered a global neuropathy instead of a pure large-fiber neuropathy." Pan at 393. Petitioner also filed

Makonahalli, et al., *Acute Small Fiber Neuropathy Following Mycoplasma Infection: A Rare Variant of Guillain-Barré Syndrome*, 15(4) J. CLINICAL NEUROMUSCULAR DISEASE 147-51 (2014) (filed as Ex. 326) ("Makonahalli"). These authors describe a single case of isolated small fiber neuropathy following pneumonia and conclude that "it is reasonable to consider this a variant of GBS." *Id.* at 148.

Based on the foregoing, I conclude that Petitioner has presented preponderant evidence that SFN is a variant of GBS.

C. *Althen* Prong One

The evidence supporting the flu vaccine as a cause of peripheral neuropathies like GBS is borne out by the inclusion of the flu vaccine/GBS relationship on the Vaccine Injury Table. 42 C.F.R. § 100.3.

In attempting to establish entitlement to a Vaccine Program award of compensation for an off-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health & Hum. Servs.*, 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires that petitioner establish by preponderant evidence that the vaccination she received caused her injury "by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* at 1278.

Under the first prong of *Althen*, a petitioner must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Proof that the proffered medical theory is reasonable, plausible, or possible does not satisfy a petitioner's burden. *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359-60 (Fed. Cir. 2019).

Several Vaccine Program cases have evaluated whether vaccination can cause small fiber neuropathy, with mixed results. Special masters have found that the flu and Tdap vaccines can cause SFN. *E.M. v. Sec'y of Health & Hum. Servs.*, No. 14-753V, 2021 WL 3477837 (Fed. Cl. Spec. Mstr. Jul 9, 2021) (finding that the flu vaccine caused Petitioner's SFN via molecular mimicry); *Swaiss*, 2019 WL 6520791 (concluding that SFN is a variant of GBS, and that the Tdap vaccine can and did cause Petitioner's condition); *Doe*, 2007 WL 3120297 (finding that the flu vaccine caused Petitioner's SFN, which was sufficiently similar to GBS to be analogous for purposes of her theory of causation). Other special masters have come to the opposite conclusion. *McGill v. Sec'y of Health & Hum. Servs.*, No. 15-1485V, 2023 WL 3813524 (Fed. Cl. Spec. Mstr. May 11, 2023) (concluding that Petitioner's vaccinations were not responsible for the development of SFN eight hours later, and that her causal theory, that vaccination resulted in dissemination of viral antigens into the dorsal root ganglia leading to the production of IFN-γ, and molecular mimicry worked in concert to create Petitioner's clinical picture, was not persuasive); (*Fantini v. Sec'y of Health & Hum. Servs.*, No 15-1332V, 2022 WL 1760730 (Fed. Cl. Spec. Mstr. May 2, 2022) (finding petitioner had not established that SFN was his correct diagnosis, and had not

presented a sound and reliable causation theory that the flu vaccine can cause SFN); *Todd v. Sec'y of Health & Hum. Servs.*, No. 15-860V, 2020 WL 727973 at *21 (Fed. Cl. Spec. Mstr. Jan. 8, 2020) (determining petitioner did not suffer from SFN).

In her expert reports, Dr. Boylan proposed that Petitioner developed a rare variant of GBS in the form of SFN, and that the mechanism by which this occurred was molecular mimicry. First Boylan Rep. at 9-10. Dr. Boylan explained the concept of molecular mimicry as occurring "when external agents share peptide or protein sequences with the cells which form the normal building blocks of the body – this is referred to as an autoimmune response." *Id.* at 9. She added that "[v]accination can induce auto-immune responses, not only through molecular mimicry but through a number of other complex immunologic mechanisms." *Id.*

The idea that a vaccine can cause an autoimmune condition through the mechanism of molecular mimicry is well established in the Vaccine Program. Special masters have accepted molecular mimicry as a sound and reliable causation theory supporting different demyelinating conditions, including GBS. *See, e.g.*, *Conte v. Sec'y of Health & Hum. Servs.*, No. 17-403V, 2020 WL 5743696, at *57 (Fed. Cl. Spec. Mstr. July 27, 2020) (noting the theory of molecular mimicry in a GBS case is "well-established and well-settled in the Vaccine Program"); *Barone v. Sec'y of Health & Hum. Servs.*, No. 11-707V, 2014 WL 6834557, at *8-9 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) (noting molecular mimicry "has been accepted in other Program cases as a reliable medical explanation for how various autoimmune conditions could develop after the receipt of different kinds of vaccinations").

Petitioners are not required to demonstrate a specific biologic mechanism that caused their disease, nor are they required to present medical literature or epidemiological studies in support of their theory. *See Kottenstette v. Sec'y of Health & Hum. Servs.*, 861 Fed. Appx. 433 (Fed. Cir. June 15, 2021) (citing *Knudsen*, 35 F.3d at 549) (reaffirming the principle that "proof of causation does not 'require identification and proof of specific biological mechanisms[.]'"); *Andreu,* 569 F.3d at 1378-79. However, a petitioner's prong one theory must be reliable.

Dr. Gibbons agreed that molecular mimicry is generally accepted as a mechanism by which the flu vaccine can cause GBS. Tr. at 221. He opined that the literature supporting autoimmune neuropathy is not strong but conceded that "a couple of antibodies [] have been reported in association with [SFN]." *Id.* at 199-200. He added, however, that the antibodies in question "were actually prevalent in a lot of people, so they really hadn't been particularly well characterized in a lot of people without neuropathy." *Id.* at 200. He opined that it is difficult to clinically link an autoimmune condition to SFN except in the cases of Sjögren's syndrome and sarcoidosis, which are thought to be associated with SFN. *Id.* He opined that the mechanism of disease in these cases is an autoimmune condition causing local reactions, not molecular mimicry. *Id.* Dr. Gibbons opined that autoimmune conditions are rarely the cause of SFN and that he has not seen data to support a vaccine as the cause of SFN. *Id.*

Petitioner has provided medical literature in support of her contention that the flu vaccine can cause GBS presenting as SFN by means of molecular mimicry. First, I note the evidence in the record supporting the premise that GBS is an immune-mediated disease process. Kuitwaard, et al., *Recurrences, vaccinations and long-term symptoms in GBS and* CIDP, 14(4) J. PERIPHERAL

NERVOUS SYSTEM 310-15, 310 (2009) ("Kuitwaard") (filed as Ex. 21); Vriesendorp at 1 ("The acute immune-mediated polyneuropathies are classified under the eponym Guillain-Barré syndrome.").

Second, there is evidence in the record supporting the contention that molecular mimicry is a sound and reliable causal theory. The article by Blank et al., states that "[d]uring the last few decades, molecular mimicry was demonstrated between self and non-self molecules that lead to an autoimmune response." Vriesendorp notes that "GBS is thought to result from an immune response to a preceding infection that cross-reacts with peripheral nerve components because of molecular mimicry…A small percentage of patients develop GBS after another triggering event such as immunization." Vriesendorp at 1.

Third, there is evidence that SFN can result from autoimmunity. Martha Kerr, *Autonomic autoimmune neuropathy is an antibody-mediated disorder*, 4(1) NEUROLOGY TODAY 4-6 (2004) (filed as Ex. 32) (describing how immune-mediated autonomic neuropathy has been induced in mice through injection with antibody-positive serum). In Levine, the authors conclude that two specific autoantibodies "are associated with sensory-predominant neuropathies or neuronopathies, and occur in up to 40% of SFN cases." Levine at 512. They note that "[a]n acute or subacute onset may define a subset of SFN that often has non-length-dependent features and may be immune in origin." *Id.* at 514.

Finally, Petitioner's medical literature supports the contention that vaccines can trigger molecular mimicry leading to autoimmune SFN. Souayah and colleagues reported five cases of paresthesias accompanied by biopsy-confirmed reduction in epidermal nerve fiber density after vaccination against rabies, varicella, or Lyme disease. Souayah, et al., *Small fiber neuropathy following vaccination for rabies, varicella or Lyme disease*, 27 VACCINE 7322-25 (2009) (filed as Ex. 28) ("Souayah"). Acknowledging that the benefits of vaccination outweigh the risks, Guimarães and colleagues point out that case reports, epidemiologic studies, and research studies "suggest a connection between several vaccines and certain autoimmune conditions." Guimarães, et al., *Vaccines, adjuvants and autoimmunity*, 100 PHARMACOLOGICAL RESEARCH 190-209, 195 (2015) (filed as Ex. 31) ("Guimarães"). Guimarães continues, saying that "[m]olecular mimicry has been suggested as a mechanism to explain an autoimmune response following influenza vaccination," and that "[d]iseases or symptoms reported after influenza vaccination include mostly neurological syndromes such as GBS." *Id.* at 196.

Viewing the record as a whole, I find that Petitioner has articulated a medically reputable theory of how the flu vaccine can cause SFN. Accordingly, the requirements of *Althen* prong one have been satisfied.

D. *Althen* Prong Three

The timing prong contains two parts. First, Petitioner must establish the "timeframe for which it is medically acceptable to infer causation" and second, they must demonstrate that the onset of the disease occurred in this period. *Shapiro v. Sec'y of Health & Hum. Servs.,* 101 Fed. Cl. 532, 542-43 (2011), *recons. denied after remand on other grounds*, 105 Fed. Cl. 353 (2012), *aff'd without op.,* 503 F. App'x 952 (Fed. Cir. 2013).

Although a temporal association alone is insufficient to establish causation, under the third prong of *Althen*, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. The special master cannot infer causation from temporal proximity alone. *See Thibaudeau v. Sec'y of Health & Hum. Servs.*, 24 Cl. Ct. 400, 403–04 (1991); *see also Grant*, 956 F.2d at 1148 ("[T]he inoculation is not the cause of every event that occurs within the ten[-]day period ... [w]ithout more, this proximate temporal relationship will not support a finding of causation." (quoting *Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983))).

1. Medically Appropriate Timeframe

With regard to a medically appropriate timeframe, Dr. Boylan opined that the five days from Petitioner's vaccination to the onset of her symptoms is an appropriate timeframe for vaccine-induced neuropathy. Tr. at 81-82. She added that when attributing neurologic syndromes to autoimmune etiologies, she prefers to see a delay of days to weeks between the antigen exposure and onset of the condition. *Id*. at 82. She also noted that it is easier to make the association with an antigenic exposure when the delay is measured in days because the proximity allows less time for potential intervening events. *Id*. Dr. Boylan concluded that five days post-vaccination is an appropriate time for autoimmune manifestation to begin. *Id*. at 83.

Dr. Boylan also opined that it is appropriate to extrapolate the Table period of three to 42 days for onset of vaccine-induced GBS symptoms to SFN. Tr. at 84. She stated that the mechanism which causes symptoms to begin from the triggering event is fundamentally the same between GBS and SFN. *Id*. Dr. Gibbons testified that up to two months was an appropriate timeframe for a vaccine to trigger a neuropathy. Tr. at 196.

Based on this evidence, I find that three days to two months is a medically appropriate timeframe for onset of SFN from which flu vaccine-causation may be reasonably inferred.

2. Petitioner's Onset

There is no dispute that Petitioner received the allegedly causal flu vaccine on September 29, 2014. Ex. 1 at 1. On November 4, 2014, 36 days after vaccination, she reported five weeks of muscle and joint pain to Dr. Ukani. Ex. 8 at 5. On November 12, 2014, Petitioner reported six weeks of numbness and tingling to Dr. Urban. Ex. 7 at 36-41. On November 20, 2014, Petitioner reported to Dr. Alboukrek that she began experiencing arthralgias and myalgias about five days after her flu vaccination, and that the tingling began shortly afterward. Ex. 4 at 9. During her testimony at the entitlement hearing, Petitioner stated that tingling and numbness in her feet started approximately five days after her flu vaccination and that it spread to her legs, arms, and hands over the following days. Tr. at 11.

I find that Petitioner's testimony that her symptoms began five days after her September 29, 2014, flu vaccination is consistent with the medical records from November 2014. Although her reports of "five weeks" and "six weeks" at her first two appointments are not precise as to the date of onset, I do not find that these records conflict with her report a few weeks later that her symptoms began five days after vaccination. I know from my experience that, when seeing a

medical provider, a patient does not always state with specificity the date on which a particular symptom began. Respondent asserts that Petitioner has failed to establish a medically acceptable timeframe for the onset of SFN but does not contest the timeline as described above. Resp. Post-Hearing Brief at 44.

The weight of the evidence supports a finding that the onset of Petitioner's muscle and joint pain occurred five days after vaccination, on or about October 4, 2014, and that her numbness and tingling began in the days immediately following. Whether the onset of Petitioner's SFN occurred with the pain on or about October 4, or with the numbness and tingling immediately following, the onset of her SFN is unquestionably within the three day to two month timeframe.

Accordingly, the requirements of the third *Althen* prong are met.

E. *Althen* Prong Two

Under *Althen's* second prong, Petitioner must "prove a logical sequence of cause and effect showing that the vaccination was the reason for the injury." *Althen*, 418 F.3d at 1278. The sequence of cause and effect must be "'logical' and legally probable, not medically or scientifically certain." *Id.* Petitioner is not required to show "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." *Id.* (omitting internal citations). *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, circumstantial evidence and reliable medical opinions may be sufficient to satisfy the second *Althen* prong. *Isaac v. Sec'y of Health & Hum. Servs.*, No. 08-601V, 2012 WL 3609993, at *25 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for rev.* denied, 108 Fed. Cl. 743 (Fed. Cl. 2013), *aff'd*, 540 Fed. Appx. 999 (Fed. Cir. 2013).

The fact that Petitioner has established that vaccination can cause SFN and that the timing prong has been met helps establish that she has also demonstrated that vaccination was a but-for cause of her condition. The Federal Circuit has provided guidance with respect to this issue.

> Evidence demonstrating petitioner's injury occurred within a medically acceptable time frame bolsters a link between the injury alleged and the vaccination at issue under the "but-for" prong of the causation analysis. *See Capizzano,* 440 F.3d at 1326 (finding medical opinions that explain how a vaccine can cause the injury alleged coupled with evidence demonstrating a close temporal relationship "are quite probative" in proving actual causation).

Pafford, 451 F.3d at 1358. *See also Contreras* (finding that there is a "logical overlap between the three *Althen* prongs, and that evidence that goes to one prong may also be probative for another prong"). 107 Fed. Cl. at 295.

I find that Petitioner has provided preponderant evidence of a logical sequence of cause and effect showing that her flu vaccine caused her to develop SFN. Petitioner's medical history supports her contention that the flu vaccine she received on September 29, 2014, caused her SFN. First, Petitioner's symptoms are consistent with the medical literature describing autoimmune

SFN. For instance, Souayah describes five cases of post-vaccination polyneuropathy presenting with paresthesias and diminished epidermal nerve fiber density, both of which Petitioner experienced. Ex. 13 at 5 (paresthesias on February 24, 2015); Ex. 13 at 11 (paresthesias on September 19, 2016); Ex. 8 at 63 (skin biopsy results showing significantly reduced epidermal nerve fiber density in Petitioner's left thigh).

Furthermore, Dr. Boylan testified that Petitioner's medical history is consistent with non-length dependent SFN because it began in her hands rather than her feet. Tr. at 70. Levine noted that the antibodies detected in their study were associated with non-length dependent neuropathic symptoms. Levine at 514. This accords with Dr. Boylan's testimony that non-length dependent symptoms are indicative of autoimmune SFN as opposed to SFN secondary to some other cause. Tr. at 71. Dr. Boylan opined that "the concordance between the constellation of symptoms in the study population and [Petitioner is] uncanny." Second Boylan Rep. at 9.

Furthermore, I did not find any evidence supporting an alternate cause of Petitioner's condition. There is no evidence in the record that Petitioner had an autoimmune disease associated with SFN or that she suffered an infection prior to developing SFN.

I also note that in several instances over the course of her medical treatment, Petitioner's treating physicians linked her condition to the flu vaccine she received on September 29, 2014. On February 9, 2015, Dr. Fernyhough made recommendations regarding over-the-counter medications due to Petitioner's "history of apparent vaccine related neuropathy syndrome." Ex. 8 at 21. On September 8, 2015, Dr. Alboukrek provided Petitioner with an exemption from the seasonal flu vaccine because she had a "severe allergic reaction" to it the previous year. Ex. 12 at 8. Dr. Alboukrek also stated with respect to Petitioner's new onset neuropathy: "I suspect that she has had some type of reaction induced by the flu shot but there is no way to prove it." Ex. 4 at 10. On October 21, 2016, Dr. Ukani's assessment was "periph[eral] neuropathy post Flu shot." Ex. 8 at 3. On July 17, 2017, Dr. Ukani wrote that "[t]he above patient is allergic to Flu shot." Ex. 12 at 21. On August 21, 2017, Dr. Buczyner's assessment was neuropathy "related to either post infectious or exposure to influenza vaccine which sounds temporally related." Ex. 11 at 8. *Id.* In this case, the opinions of Petitioner's treating physicians provide persuasive evidence in support of her position that the flu vaccine did cause her condition. *Pafford*, 451 F.3d at 1358.

Viewing the medical record as a whole, I find that Petitioner has presented preponderant evidence in support of the second *Althen* prong.

VIII. Conclusion

Upon careful evaluation of all the evidence submitted in this matter, including the medical records, the experts' opinions and medical literature, and the testimony, I conclude that Petitioner has provided preponderant evidence in support of her claim that the flu vaccine caused her to develop SFN. She is therefore entitled to compensation under the Vaccine Act. A damages order will issue shortly.

IT IS SO ORDERED.

s/ Katherine E. Oler
Katherine E. Oler
Special Master